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MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE FORTY-THIRD.

LONDON:
LONGMAN, GREEN, LONGMAN, AND ROBERTS,
PATERNOSTER ROW.

1860.

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MEDICO-CHIRURGICAL TRANSACTIONS.

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SECOND SERIES.

VOLUME THE TWENTY-FIFTH.

MDCCCV.

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PATERNOSTER ROW.

1860.

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OF LONDON.

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The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed.

OCTOBER 1860.

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Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *James Abercrombie, M.D., Cape of Good Hope.

1846 *John Abercrombie, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.

1851 *Henry Wentworth Acland, M.D., F.R.S., Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Elias Acosta, M.D., New York, U.S.

1842 William Acton, 17, Queen Anne street, Cavendish square. *Trans.* 1.

Elected

1851 John Adams, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 4, St. Helen's place, Bishopsgate street. *Trans.* 2.

1852 William Adams, Surgeon to the Royal Orthopædic Hospital; Lecturer on Anatomy and Surgery at the Grosvenor place School of Anatomy and Medicine; 5, Henrietta street, Cavendish square. *Trans.* 2.

1837 *Ralph Fawsett Ainsworth, M.D., Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1819 George Frederick Albert.

1839 Rutherford Alcock, K.C.T., K.T.S., H.M.'s Envoy Extraordinary and Consul-General in Japan. *Trans.* 1.

1826 James Alderson, M.D., F.R.S., Senior Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. *Trans.* 3.

1843 Charles James Berridge Aldis, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester terrace, Chester square.

1850 Charles Revans Alexander, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.

1836 Henry Ancell, 3, Norfolk crescent, Oxford square. C. 1847-8. *Trans.* 2.

1820 Thomas Andrews, M.D., Norfolk, Virginia.

1819 Professor Antommarchi, Florence.

1819 †James Moncrieff Arnott, F.R.S., Surgeon in Ordinary to H.R.H. the Prince Consort; 2, New Burlington street. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †John Ashburner, M.D., F.L.S., 7, Hyde Park place, Cumberland gate. C. 1821, 1830-1.

1851 Thomas John Ashton, Surgeon to the Blenheim Free Dispensary; 31, Cavendish square.

Elected

1825 †Benjamin Guy Babington, M.D., F.R.S., Physician to the Asylum for Deaf and Dumb, Consulting Physician to the German Hospital, and to the City of London Hospital for Diseases of the Chest; 31, George street, Hanover square. C. 1829. V.P. 1845-6. T. 1848. *Trans.* 2.

1846 Cornelius Metcalfe Stuart Babington, F.R.C.P., Physician to Queen Charlotte's Lying-in Hospital, and Assistant-Physician to the Hospital for Sick Children; 29, Hertford street, May fair. C. 1859-60.

1820 *John H. Badley, Dudley, Worcestershire.

1838 Francis Badgley, M.D., Holyrood House, Great Malvern.

1840 William Bainbridge, late of Kingston, Surrey.

1836 Andrew Wood Baird, M.D., Physician to the Dover Hospital; Dover, Kent.

1851 *Alfred Baker, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; Cannon street, Birmingham.

1839 †Thomas Graham Balfour, M.D., F.R.S., *Vice-President*, Deputy Inspector-General of Hospitals; 10, Sumner place, Onslow square, Brompton. C. 1852-3. *Trans.* 2.

1848 Edward Ballard, M.D., Medical Officer of Health for Islington; 7, Compton terrace, Upper street, Islington. *Trans.* 1.

1849 Thomas Ballard, 10, Southwick place, Hyde park.

1837 William Baly, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Assistant-Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 19, Queen Anne street, Cavendish square. C. 1845-6. L. 1847. S. 1848-9. V.P. 1855-6. T. 1859. *Trans.* 1.

1847 Andrew Whyte Barclay, M.D., *Secretary*, Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Medical Officer of Health for Chelsea; 23a, Bruton street, Berkeley square. S. 1857-9. *Trans.* 2.

1848 Edgar Barker, 9, Oxford square, Hyde park.

Elected

1833 †Thomas Alfred Barker, M.D., *Treasurer*, Senior Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 71, Grosvenor street. C. 1844-5. V.P. 1853-4. *Trans.* 6.

1843 Thomas Herbert Barker, M.D., Harpur place, Bedford.

1847 George Hilaro Barlow, M.D., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; Physician to the Magdalen Hospital; 5, Union street, Southwark. C. 1859.

1840 Benjamin Barrow, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.

1859 Richard Barwell, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, the Charing Cross Hospital; 22, Old Burlington street.

1844 William Richard Basham, M.D., Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Grosvenor place. S. 1852-4. C. 1860. *Trans.* 2.

1860 Adam Bealey, M.D., M.A. Camb., Physician to the St. Pancras Dispensary; 27, Tavistock square.

1841 George Beaman, M.D., 5, Lancaster place, Strand.

1856 Amos Beardsley, Ulverstone, Lancashire.

1836 William Beaumont, Professor of Surgery in the University of King's College, Toronto, Upper Canada. *Trans.* 2.

1840 Charles Beevor, 41, Upper Harley street.

1858 William Chapman Begley, M.D., Middlesex County Lunatic Asylum; Hanwell.

1819 †Thomas Bell, F.R.S., F.L.S., Professor of Zoology in King's College, London; Surgeon-Dentist to, and Lecturer on the Anatomy and Diseases of the Teeth at, Guy's Hospital, and President of the Linnean Society; 17, New Broad street, City. C. 1832-3. V.P. 1854. *Trans.* 1.

1847 James Henry Bennet, M.D., Physician-Accoucheur to the Royal Free Hospital.

1845 Edwin Unwin Berry, 7, James street, Covent garden.

1820 Stephen Bertin, Paris.

Elected

1815 †Archibald Billing, M.D., F.R.S., late Senior Physician to the London Hospital; Member of the Senate, and Examiner in Medicine at the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.

1827 William Birch, Barton-under-Needwood, Staffordshire. *Trans.* 2.

1850 James Bird, M.D., Lecturer on Military Surgery at St. Mary's Hospital Medical School, 27, Hyde park square.

1855 Peter Hinckes Bird, F.L.S., 1, Norfolk square, Hyde park.

1856 William Bird, Surgeon to the West of London Hospital, and the St. George's and St. James's Dispensary; 11, George street, Hanover square.

1849 Edmund Lloyd Birkett, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square.

1851 George Birkett, M.D., Lecturer on Medical Jurisprudence at the Charing Cross Hospital; Northumberland House, Green lanes, Stoke Newington.

1851 John Birkett, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. *Trans.* 4.

1846 Hugh Birt, 12, High street, Portsmouth.

1843 Patrick Black, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square. C. 1856.

1847 George C. Blackman, M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.

1839 Richard Blagden, Surgeon Extraordinary to H.M. the Queen, and Surgeon in Ordinary to H.R.H. the Duchess of Kent; 7, Percy place, Walcot, Bath. C. 1847-8. *Trans.* 1.

1840 Peyton Blakiston, M.D., F.R.S., St. Leonard's-on-Sea.

1845 Henry Blenkinsop, Senior Surgeon to the Warwick Dispensary; Jury street, Warwick.

1823 Louis Henry Bojanus, M.D., Wilna.

1846 Peter Bossey, 4, Broadwater road, Worthing, Sussex.

Elected

1846 John Ashton Bostock, Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia.

1841 William Bowman, F.R.S., Surgeon to King's College Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. *Trans.* 3.

1857 William Brinton, M.D., Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 20, Brook street, Grosvenor square.

1851 Bernard Edward Brodhurst, Assistant-Surgeon to the Royal Orthopædic Hospital, and Senior Surgeon to the Hon. Artillery Company; 20, Grosvenor street. *Trans.* 2; *Pro.* 1.

1813 †Sir Benjamin Collins Brodie, Bart., D.C.L., F.R.S., President of the Royal Society, Serjeant-Surgeon to H.M. the Queen, Surgeon in Ordinary to H.R.H. the Prince Consort, Foreign Correspondent of the Institute of France, and Foreign Associate of the Imperial Academy of Medicine of Paris; 14, Savile row, Burlington gardens. C. 1814-15, 1818-19, 1821-2, 1835-6, 1841-2, 1849. V.P. 1816-17. P. 1839-40. *Trans.* 11.

1844 Charles Brooke, M.A., F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 16, Fitzroy square. C. 1855.

1848 William Philpot Brookes, M.D.

1854 *Henry Brown, Surgeon to H.M. the Queen, H.R.H. the Prince Consort, and the Royal Household; Windsor.

1857 *Robert Brown, Surgeon to the Carlisle Dispensary; 4, Devonshire street, Carlisle.

1860 Charles Edouard Brown-Séquard, M.D., F.R.S., Physician to the National Hospital for the Paralysed and Epileptic; 81, Wimpole street.

1851 Alexander Browne, M.D., Twynholm, Kirkcudbright.

1860 Thomas Bryant, Assistant-Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 40, Wellington street, Southwark.

Elected

1855 Walter John Bryant, L.R.C.P., Edinb., 7, Bathurst street, Hyde park gardens.

1823 B. Bartlet Buchanan, M.D.

1843 John Charles Bucknill, M.D., Medical Superintendent of the Devon County Lunatic Asylum, Exminster, Devonshire.

1839 George Budd, M.D., F.R.S., Professor of Medicine in King's College, London; Physician to King's College Hospital; Consulting Physician to the Seamen's Hospital Ship 'Dreadnought,' and to the Blenheim Free Dispensary; 20, Dover street, Piccadilly. C. 1846-7. V.P. 1857. *Trans.* 5.

1839 Thomas Henry Burgess, M.D., Portsmouth.

1853 Patrick Burke, 13, Upper Montagu street, Montagu square.

1854 Philip Burrowes, Surgeon to the London City Mission, and Assistant-Surgeon to the Hospital for Women; 23, Gloucester crescent north, Hyde park.

1833 †George Burrows, M.D., F.R.S., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.

1820 Samuel Burrows.

1837 George Busk, F.R.S., F.L.S., Examiner in Comparative Anatomy at the University of London, Surgeon to the Seamen's Hospital Ship 'Dreadnought;' 15, Harley street, Cavendish square. C. 1847-8. V.P. 1855. *Trans.* 4.

1818 John Butter, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Plymouth.

1851 *William Cadge, Surgeon to the Norfolk and Norwich Hospital; All Saints, Norwich. *Trans.* 1.

1851 Thomas Callaway, India.

1852 *George Canney, Bishop-Auckland, Durham.

1847 John Burford Carlill, M.D., Surgeon-Accoucheur to the Newman street Lying-in Institution; 57, Berners street.

1825 Harry W. Carter, M.D., F.R.S.E., Consulting Physician to the Kent and Canterbury Hospital; Ashford, Kent.

Elected

1853 Robert Brudenell Carter, Newthorpe, Nottinghamshire.

1820 †Samuel Cartwright, F.R.S., F.L.S., Nizell's House, near Tunbridge, Kent.

1845 Samuel Cartwright, Jun., Surgeon-Dentist to King's College Hospital; 32, Old Burlington street. C. 1860.

1839 William Cathrow, 42, Weymouth street, Portland place. C. 1860.

1845 William Oliver Chalk, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park [40, Marylebone road].

1844 Thomas King Chambers, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Lock Hospital; 1, Hill street, Berkeley square. *Trans.* 1.

1859 Frank Chance, M.B., Physician to the Blenheim Free Dispensary; 51, Wimpole street, Cavendish square.

1849 Frederick Chapman, Richmond green, Surrey.

1837 Henry Thomas Chapman, 16, Lower Seymour street, Portman square. C. 1858.

1852 George Borlase Childs, Surgeon-in-Chief to the City Police Force, and Surgeon to the Metropolitan Free Hospital; 11, Finsbury place South.

1842 William Dingle Chowne, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 8, Connaught place West, Hyde park. C. 1853-4.

1860 Andrew Clark, M.D., Assistant-Physician to the London Hospital, and Lecturer on Physiology at the London Hospital Medical College; 23, Montague place, Russell square.

1839 Frederick le Gros Clark, Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P. 1855-6. *Trans.* 3.

Elected

1845 John Clark, M.D., Staff Surgeon, 1st Class. Canada.

1848 John Clarke, M.D., Physician to the British Lying-in Hospital, and Assistant-Physician to the General Lying-in Hospital; 42, Hertford street, Mayfair.

1850 Josiah Clarkson, New Hall street, Birmingham. *Trans.* 1.

1842 Oscar Moore Passey Clayton, 87, Harley street.

1853 Joseph Thomas Clover, 3, Cavendish place, Cavendish square.

1857 Charles Coates, F.R.C.P. Edinb., Physician to the Bath General Hospital; 10, Circus, Bath.

1851 Edward Cock, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; Consulting Surgeon to the Asylum for Deaf and Dumb; 13, St. Thomas's street, Southwark. C. 1857. *Trans.* 3.

1850 Daniel Whitaker Cohen, M.D., Headley grove, near Dorking.

1835 *William Colborne, Chippenham, Wiltshire.

1818 Robert Cole, F.L.S., Holybourne, Hampshire.

1855 Frederick Collins, M.D., Medical Officer of Health for Wanstead; Wanstead, Essex.

1828 John Conolly, M.D., D.C.L., Consulting Physician to the Middlesex County Lunatic Asylum, Hanwell.

1840 *William Robert Cooke, Burford, Oxfordshire.

1819 George Cooper, Brentford, Middlesex.

1841 George Lewis Cooper, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860.

1843 William White Cooper, Surgeon-Oculist in Ordinary to H.M. the Queen; Senior Surgeon to the North London Eye Infirmary; and Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Mary's Hospital; 19, Berkeley square. C. 1858-9.

1854 Charles Thomas Coote, M.D., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital; 1, Gloucester place, Hyde park.

1841 Holmes Coote, Assistant-Surgeon to St. Bartholomew's Hospital, and to the Royal Orthopædic Hospital; 26, New Bridge street, Blackfriars. S. 1853-4. *Trans.* 1.

Elected	
1835	George Ford Copeland, Cheltenham.
1822	†James Copland, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital, King's Cross; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street. C. 1831. V.P. 1838-9. P. 1853-4.
1847	John Rose Cormack, M.D., F.R.S.E., 5, Bedford square.
1860	*Thomas Charles Steuart Corry, M.D., Surgeon to the Belfast General Dispensary: 1, Victoria street, Belfast.
1839	*Charles Cæsar Corsellis, M.D., F.L.S., Benson, Oxon.
1853	William Gillett Cory, M.D., Burgh Heath, Sutton, Surrey.
1847	Richard Payne Cotton, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly.
1828	†William Coulson, Senior Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Consulting Surgeon to the German Hospital; 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.
1860	John Couper, Demonstrator of Anatomy at the London Hospital; 9, Finsbury Circus.
1841	Mervyn Archdall Nott Crawford, M.D., Wiesbaden. C. 1853-4.
1847	George Critchett, Senior Assistant-Surgeon to, and Lecturer on Surgery at, the London Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 46, Finsbury square. *Trans.* 1.
1837	John Farrar Crookes, Harewell, near Faversham, Kent.
1849	*William Edward Crowfoot, Beccles, Suffolk.
1851	James Cameron Cumming, M.D., 1, Cadogan place, Sloane street.
1846	Henry Curling, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.
1837	Thomas Blizard Curling, F.R.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; Examiner in Surgery at the University of London; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. *Trans.* 13, *Pro.* 1.

Elected

1847 John Edmund Currey, M.D., Lismore, County Waterford.

1836 George Cursham, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, and to the Female Orphan Asylum; and Inspector of Anatomy for the Provinces; 55, Victoria street, Westminster. S. 1842-7. C. 1850-1. V.P. 1855. T. 1856-8. *Trans.* 1.

1822 Christopher John Cusack, Chateau d'Eu, France.

1852 Thomas Cutler, M.D., Acting Physician at the Spa Waters; Spa, Belgium.

1828 Adolphe Dalmas, M.D., Paris.

1836 *James Stock Daniel, Ramsgate, Kent.

1820 †George Darling, M.D., 6, Russell square. C. 1841-2.

1848 Henry Daubeny.

1846 Frederick Davies, Surgeon to the Northern Dispensary; 19, Upper Gower street, Bedford square.

1818 †Henry Davies, M.D., Consulting-Physician to the British Lying-in Hospital; 6, Duchess street, Portland place. C. 1827-8. V.P. 1848-9.

1847 John Davies, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.

1853 Robert Coker Nash Davies, Rye, Sussex.

1852 William Davies, M.D., Senior Physician to the Bath United Hospital; 10, Gay street, Bath.

1852 John Hall Davis, M.D., Physician to the Royal Maternity Charity; Physician-Accoucheur to the St. George's and St. James's Dispensary; and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 11, Harley street, Cavendish square.

1820 †Thomas Davis, 28, Spring gardens. C. 1837, 1843.

1818 James Dawson, Liverpool.

1847 George Edward Day, M.D., F.R.S., Chandos Professor of Anatomy, and Examiner in Medicine in the University of St. Andrew's.

1858 Teofilo Delima, M.D., Caracas, Venezuela, South America.

1846 *Samuel Best Denton, M.D., Ivy Lodge, Hornsea, East Riding, Yorkshire.

Elected

1859 William Howship Dickinson, M.B., Medical Registrar at St. George's Hospital; 9, Chesterfield street, May fair. *Trans.* 1.

1844 Robert Dickson, M.D., F.L.S., Physician to the Scottish Hospital, and to the British Orphan Asylum, Clapham; 16, Hertford street, May fair. C. 1860.

1839 †James Dixon, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman square. L. 1849-55. V.P. 1857-8. *Trans.* 4.

1845 John Dodd.

1857 Archibald Douglas, M.D., 8, Clifton place, Sussex square, Hyde park.

1853 Robert Druitt, M.R.C.P., Medical Officer of Health for St. George's, Hanover square; 37, Hertford street, May fair. *Trans.* 2.

1846 John Drummond, Deputy-Inspector of Fleets and Hospitals. *Trans.* 1.

1845 George Duff, M.D., High street, Elgin.

1845 Edward Willson Duffin, 14, Langham place. *Trans.* 1.

1833 Robert Dunn, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1843 Christopher Mercer Durrant, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 Henry Sumner Dyer, M.D., 37, Bryanston sq. C. 1854-5.

1836 James William Earle, late of Norwich.

1824 George Edwards.

1823 Charles Chandler Egerton, Kendall Lodge, Epping.

1848 George Viner Ellis, Professor of Anatomy in University College, London; University College, Gower street. *Trans.* 2.

1854 *James Ellison, M.D., Surgeon in Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1835 William England, M.D., Ipswich, Suffolk.

1842 John Erichsen, Professor of Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. *Trans.* 2.

Elected

1836 George Fabian Evans, M.D., Physician to the General Hospital, Birmingham.

1815 *Griffith Francis Dorsett Evans, M.D., St. Mary's, Bedford. C. 1838.

1845 William Julian Evans, M.D., Pinner, Middlesex.

1858 Randle Wilbraham Falconer, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

1844 Arthur Farre, M.D., F.R.S., Professor of Midwifery in King's College, London, and Physician for the Diseases of Women and Children, to King's College Hospital; 12, Hertford street, May fair. C. 1857.

1831 Robert Ferguson, M.D., Physician Extraordinary to H.M. the Queen, and Consulting Physician to King's College Hospital; 125, Park street, Grosvenor square. C. 1839. V.P. 1847.

1841 William Fergusson, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince Consort; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; Examiner in Surgery at the University of London; 16, George street, Hanover square. C. 1849-50. *Trans.* 4.

1852 *Alfred George Field, Surgeon to St. Mary's Hospital, Brighton; 28, Old Steine, Brighton.

1849 George Tupman Fincham, M.D., Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital; 2, Eccleston terrace south, Eccleston square.

1836 Sir John William Fisher, Surgeon-in-Chief to the Metropolitan Police Force; 5, Grosvenor gate. C. 1843-4.

1860 Thomas George Fitzgerald, Staff-Surgeon; 6, Whitehall yard.

1842 Thomas Bell Elcock Fletcher, M.D., Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.

1859 William Henry Flower, Assistant-Surgeon to the Middlesex Hospital; 32, Queen Anne street, Cavendish square. *Trans.* 1.

Elected

1848 John Gregory Forbes, Surgeon to the Metropolitan Convalescent Institution; 9, Devonport street, Hyde park. *Trans.* 2.

1852 †John Cooper Forster, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Royal Infirmary for Children, &c.; 11, Wellington street, Southwark. *Pro.* 1.

1859 Edward Long Fox, M.B., Physician to the Bristol Royal Infirmary; 10, Berkeley square, Bristol.

1858 *Wilson Fox, M.D., Physician to the North Staffordshire Infirmary; Newcastle-under-Lyme, Staffordshire.

1856 John F. France, Lecturer on Ophthalmic Surgery at Guy's Hospital, and Surgeon to the Eye Infirmary attached to the Hospital; 24, Bloomsbury square.

1816 John W. Francis, M.D., LL.D., Professor of Materia Medica in the University of New York, U.S.

1841 John Christopher Augustus Franz, M.D., 11, Old Steine, Brighton.

1843 Patrick Fraser, M.D., Physician to the London Hospital, and to the London Dispensary; 63, Grosvenor street.

1836 John George French, Surgeon to the St. James's Infirmary; 41, Great Marlborough street. C. 1852-3.

1849 Robert Temple Frere, M.A., F.R.C.P., Physician-Accoucheur to the Middlesex Hospital; 9, Queen street, May fair.

1846 Henry William Fuller, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 13, Manchester square. *Trans.* 2.

1815 *George Frederick Furnivall, Medical Attendant of Great Foster House Asylum for Lunatics; Egham, Surrey.

1854 Alfred Baring Garrod, M.D., F.R.S., Examiner in Materia Medica at the University of London; Professor of Materia Medica, Therapeutics, and Clinical Medicine in University College, London, and Physician to University College Hospital; 84, Harley street, Cavendish square. *Trans.* 8.

Elected

1857 GEORGE GREEN GASCOYEN, Assistant-Surgeon to the Lock Hospital, and Lecturer on Descriptive and Surgical Anatomy in the St. Mary's Hospital Medical School; 25, Oxford terrace, Hyde park. *Trans.* 1.

1851 GEORGE GASKOIN, 3, Westbourne park.

1819 HENRY GAULTER.

1848 JOHN GAY, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

1821 *RICHARD FRANCIS GEORGE, Senior Surgeon to the Bath General Hospital; 10, Royal Crescent, Bath.

1854 BERNARD GILPIN, Belle Vue House, Ulverstone, Lancashire.

1858 BENJAMIN GODFREY, M.D., Carlton House, Enfield, Middlesex.

1851 STEPHEN JENNINGS GOODFELLOW, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 5, Savile row, Burlington gardens. *Trans.* 1

1818 JAMES ALEXANDER GORDON, M.D., C.B., F.R.S., Parham House, Dorking. C. 1828. V.P. 1829. *Trans.* 1.

1851 PETER YEAMES GOWLLAND, Assistant-Surgeon to the London Hospital, and Surgeon to St. Mark's Hospital; 34, Finsbury square.

1844 JOHN GRANTHAM, Crayford, Kent.

1850 HENRY GRAY, F.R.S., Lecturer on Anatomy at St. George's Hospital Medical School, and Surgeon to the St. George's and St. James's Dispensary; 8, Wilton street, Grosvenor place. *Trans.* 2.

1846 GEORGE THOMPSON GREAM, M.D., 2, Upper Brook street, Grosvenor square.

1816 JOSEPH HENRY GREEN, D.C.L., F.R.S., President of the Medical Council; Consulting Surgeon to St. Thomas's Hospital; Hadley, Middlesex. C. 1820. V.P. 1830. *Trans.* 1.

1843 ROBERT GREENHALGH, M.D., Consulting Physician-Accoucheur to the St. John's Wood Dispensary; 11, Upper Woburn place, Tavistock square.

Elected

1860 Edward Headlam Greenhow, M.D., Lecturer on Public Health at St. Thomas's Hospital, and Physician to the Western General Dispensary; 77, Upper Berkeley street, Portman square.

1814 John Grove, M.D., Salisbury.

1852 John Grove, West Hill, Wandsworth, Surrey.

1860 Henri Gueneau de Mussy, M.D., 4, Cavendish place, Cavendish square.

1849 William Withey Gull, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, and Member of the Senate of the University of London; 8, Finsbury square. *Trans.* 2.

1837 James Manby Gully, M.D.; Great Malvern, Worcestershire.

1859 Theophilus Miller Gunn; 40, York place, Portman square.

1819 John Gunning, C.B., Inspector-General of Hospitals; Paris. C. 1823.

1854 Samuel Osborne Habershon, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; 22, Wimpole street, Cavendish square. *Trans.* 1.

1849 Hammett Hailey, Newport Paguell, Bucks.

1845 Sir John Hall, M.D., K.C.B., Inspector-General of Hospitals.

1848 Alexander Halley, M.D., F.G.S., 7, Harley street, Cavendish square.

1819 †Thomas Hammerton, 112, Piccadilly. C. 1829–30.

1838 Henry Hancock, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital, and Surgeon to the Royal Westminster Ophthalmic Hospital; 37, Harley street, Cavendish square. C. 1851.

1849 *Richard James Hansard, Surgeon to the Radcliffe Infirmary; 5, Broad street, Oxford.

1848 *George Harcourt, M.D., Chertsey, Surrey.

1836 John Fosse Harding, Sandford House, Highbury New Park. C. 1858-9.

Elected

1856 Charles John Hare, M.D., Physician to University College Hospital; 41, Brook street, Grosvenor square.

1858 William Warwick Harkness, late Demonstrator of Anatomy at the London Hospital Medical College; 9, Finsbury circus.

1857 George Harley, M.D., F.C.S., Professor of Medical Jurisprudence in University College, London; 77, Harley street, Cavendish square.

1859 Francis Harris, M.D., Demonstrator of Pathological Anatomy at St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 11, New Cavendish street, Portland place.

1846 John Harrison, 2, the Court yard, Albany.

1843 Thomas Sunderland Harrison, M.D., F.L.S., Innox Hill House, Frome, Somersetshire.

1841 William Harvey, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1855 Alfred Haviland, Surgeon to the Bridgewater Infirmary; Bridgewater, Somerset.

1828 †Cæsar Henry Hawkins, F.R.S., Surgeon Extraordinary to H.M. the Queen, and Senior Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 Charles Hawkins, Inspector of Anatomy, Consulting Surgeon to Queen Charlotte's Hospital; 22, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. *Trans.* 2.

1848 Thomas Hawksley, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 26, George street, Hanover square.

1860 Henry Howard Hayward, 56, Queen Anne street, Cavendish square.

1820 Thomas Emerson Headlam, M.D., Consulting Physician to the Newcastle Infirmary, Newcastle-upon-Tyne.

1848 *James Newton Heale, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1850 George Heaton, M.D., Boston, U.S.

Elected

1829 THOMAS HEBERDEN, M.D., 43, Park street, Grosvenor square.

1844 JOHN HENNEN, M.D. L. 1848-50.

1849 AMOS HENRIQUES, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

1848 MITCHELL HENRY, Surgeon to, and Lecturer on Medical Jurisprudence at, the Middlesex Hospital; Surgeon to the North London Eye Infirmary; 5, Harley street, Cavendish square. *Trans.* 2.

1821 VINCENT HERBERSKI, M.D., Professor of Medicine in the University of Wilna.

1843 PRESCOTT GARDNER HEWETT, Assistant-Surgeon to St. George's Hospital; 1, Chesterfield street, May fair. C. 1859. *Trans.* 7.

1855 GRAILY HEWITT, M.D., Physician to the British Lying-in Hospital; Lecturer on Midwifery and the Diseases of Women and Children, and on Comparative Anatomy at St. Mary's Hospital; 17, Radnor place, Hyde park.

1853 THOMAS HEWLETT, Surgeon to Harrow School; Harrow, Middlesex. *Trans.* 1.

1841 *NATHANIEL HIGHMORE, Sherborne, Dorsetshire.

1854 THOMAS HILLIER, M.D., Medical Officer of Health for St. Pancras, and Assistant-Physician to the Hospital for Sick Children; 21, Upper Gower street.

1842 WILLIAM AUGUSTUS HILLMAN, Senior Assistant-Surgeon to the Westminster Hospital; 1, Argyll street, Regent street. C. 1858-9.

1841 †JOHN HILTON, F.R.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting-Surgeon to the Royal General Dispensary, St. Pancras; Professor of Anatomy and Surgery at the Royal College of Surgeons; 10, New Broad street, City. C. 1851. *Trans.* 3.

1859 FRANCIS HIRD, Assistant-Surgeon to, and Lecturer at, the Charing Cross Hospital; 17, Clifford street, Bond street.

1840 THOMAS HODGKIN, M.D., Consulting Physician to the Hospital for Diseases of the Skin, and Member of the Senate of the University of London; 35, Bedford square. C. 1842-3. *Trans.* 6.

Elected

1813 JOSEPH HODGSON, F.R.S.. 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.

1843 LUTHER HOLDEN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital, and Surgeon to the Metropolitan Dispensary; 54, Gower street, Bedford square. C. 1859.

1814 †SIR HENRY HOLLAND, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince Consort; 25, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1856 TIMOTHY HOLMES, Curator of the Pathological Museum of St. George's Hospital, and Assistant-Surgeon to the Hospital for Sick Children; 22, Queen street, May fair. *Trans.* 2.

1846 BARNARD WIGHT HOLT, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 5, Parliament street.

1846 CARSTEN HOLTHOUSE, Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's gate, St. James's park.

1853 WILLIAM CHARLES HOOD, M.D., Resident Physician and Medical Superintendent of Bethlem Hospital. *Trans.* 1.

1828 *EDWARD HOWELL, M.D., Senior Consulting Physician to the Swansea Infirmary; 2, South Hill place, Swansea, Glamorganshire.

1857 JOHN WHITAKER HULKE, Assistant-Surgeon to King's College Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington st. *Trans.* 2.

1857 EDWARD CHARLES HULME, Surgeon to the Central London Ophthalmic Hospital, Surgeon to the Blenheim Free Dispensary, and Examining Surgeon to the Marine Society; 19, Gower street, Bedford square.

1844 EDWIN HUMBY, 1, Windsor terrace, Maida Hill.

1855 GEORGE MURRAY HUMPHRY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital, and Lecturer on Anatomy in the Cambridge University Medical School; Cambridge. *Trans.* 2.

Elected

1840 Henry Hunt, M.R.C.P., 68, Brook street, Hanover square. C. 1857. *Trans.* 2.

1849 Edward Law Hussey, Surgeon to the Radcliffe Infirmary, St. Aldate's, Oxford. *Trans.* 1.

1856 Jonathan Hutchinson, Assistant-Surgeon to the London Hospital, and Surgeon to the Metropolitan Free Hospital; 14, Finsbury circus. *Pro.* 2.

1820 William Hutchinson, M.D.

1840 Charles Hutton, M.D., Physician to the General Lying-in Hospital, and to the Royal Infirmary for Children and Women; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 William Edmund Image, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 Cornelius Inglis, M.D.

1826 William Ingram, Midhurst, Sussex.

1845 *Henry Jackson, Senior Surgeon to the Sheffield General Infirmary; St. James's row, Sheffield, Yorkshire.

1841 Paul Jackson, 24, Wimpole street, Cavendish square.

1841 Maximilien Morris Jacobovics, M.D., Vienna.

1825 John B. James, M.D.

1847 *William Withall James, Surgeon to the Devon and Exeter Hospital; Exeter, Devonshire.

1844 Samuel John Jeaffreson, M.D., Physician to the Warneford Hospital, and Warwick Dispensary; Leamington, Warwickshire.

1839 Julius Jeffreys, F.R.S., Kingston, Surrey.

1840 *George Samuel Jenks, M.D., 18, Circus, Bath.

1851 William Jenner, M.D., Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Physician to the Hospital for Sick Children; 8, Harley street, Cavendish square. *Trans.* 3.

1848 Athol Archibald Wood Johnson, Lecturer on Anatomy and Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37, Albemarle street, Piccadilly. *Trans.* 1.

Elected

1851 Edmund Charles Johnson, M.D., Corresponding Member of the Imperial Society of Florence; 6, Savile row.

1821 Sir Edward Johnson, M.D., Weymouth, Dorsetshire.

1847 George Johnson, M.D., Professor of Materia Medica and Therapeutics in King's College, London, and Physician to King's College Hospital; 11, Savile row, Burlington gardens. *Trans.* 5.

1837 Henry Charles Johnson, *Vice-President,* Surgeon to St. George's Hospital; 6, Savile row, Burlington gardens. C. 1850-1.

1844 †Henry Bence Jones, M.D., F.R.S., Physician to St. George's Hospital; 31, Brook street, Grosvenor square. C. 1855-6. *Trans.* 11.

1835 Henry Derviche Jones, 23, Soho square. C. 1854-5.

1853 Thomas Wharton Jones, F.R.S., Professor of Ophthalmic Surgery in University College, London, and Ophthalmic Surgeon to University College Hospital; 35, George street, Hanover square. *Trans.* 1.

1837 Thomas William Jones, M.D., 19, Finsbury pavement. C. 1858.

1859 William Price Jones, M.D., Surbiton, Kingston.

1829 *George Charles Julius, Richmond, Surrey.

1816 *George Hermann Kauffmann, M.D., Hanover.

1848 *Daniel Burton Kendell, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.

1847 Alfred Keyser, 21, Norfolk crescent, Oxford square.

1857 Henry Walter Kiallmark, late Staff Surgeon, 2d class, attached to the Ottoman Army; 46, Prince's square, Westbourne grove.

1839 *David King, M.D., Medical Officer of Health for Eltham; Eltham, Kent.

1851 John Abernethy Kingdon, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City.

1858 William Senhouse Kirkes, M.D., Assistant-Physician to St. Bartholomew's Hospital; 2, Lower Seymour street, Portman square. *Trans.* 1.

Elected

1840 SAMUEL ARMSTRONG LANE, Surgeon to St. Mary's Hospital, and Consulting Surgeon to the Lock Hospital; 1, Grosvenor place. C. 1849-50.

1855 JAMES ROBERT LANE, Surgeon to, and Lecturer on Anatomy and Physiology at, St. Mary's Hospital; and Surgeon to the Lock and St. Mark's Hospitals; 1, Grosvenor place.

1841 *CHARLES LASHMAR, M.D., 83, North End, Croydon, Surrey.

1816 G. E. LAWRENCE.

1809 †WILLIAM LAWRENCE, F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital, and Surgeon to Bridewell, and Bethlem Hospital, Foreign Associate of the Imperial Academy of Medicine of Paris; 18, Whitehall place. S. 1813-7. V.P. 1818-9. T. 1821-6. P. 1831-2. C. 1820, 1833-4, 1842-3. *Trans.* 18.

1840 THOMAS LAYCOCK, M.D., F.R.S.E., Professor of the Practice of Medicine in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.

1843 *JESSE LEACH, Moss Hall, Heywood, near Bury, Lancashire.

1822 JOHN JOSEPH LEDSAM, M.D., 17, Esplanade, Scarborough, Yorkshire.

1823 †HENRY LEE, M.D., F.L.S., Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.

1843 HENRY LEE, Surgeon to King's College Hospital, and Senior Surgeon to the Lock Hospital; 9, Savile row, Burlington gardens. C. 1856-7. *Trans.* 5. *Pro.* 1.

1822 †ROBERT LEE, M.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 22.

1836 FREDERICK LEIGHTON, M.D., Frankfort-on-the-Maine.

1854 HANANEL DE LEON, M.D., 6, Victoria terrace, Bedford.

1856 DAVID LEWIS, M.D., Physician to the Royal Society of Ancient Britons' Schools.

Elected

1847 Sir John Liddell, M.D., C.B., F.R.S., Hon. Physician to H.M. the Queen, Director-General of the Medical Department of the Navy; Admiralty, Somerset House.

1806 John Lind, M.D.

1845 William John Little, M.D., Physician to the London Hospital; 34, Brook street, Grosvenor square.

1819 Robert Lloyd, M.D.

1824 †Eusebius Arthur Lloyd, Surgeon to St. Bartholomew's and Christ's Hospitals; 14, Bedford row. S. 1827-8. V.P. 1838. C. 1843-4. *Trans.* 2.

1820 J. G. Locher, M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †Sir Charles Locock, Bart., M.D., First Physician-Accoucheur to H.M. the Queen, and Consulting Physician to the General Lying-in Hospital; Member of the Senate of the University of London; 26, Hertford street, May fair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

1852 Charles Lodge, M.D.

1846 Henry Thomas Lomax, Surgeon to the County Police; St. Mary's grove, Stafford.

1860 Thomas Longmore, Deputy Inspector-General and Professor of Clinical and Military Surgery; New Army Medical School, Chatham.

1836 Joseph S. Löwenfeld, M.D., Berbice.

1852 James Luke, F.R.S., Senior Surgeon to the London Hospital, and Surgeon to St. Luke's Hospital for Lunatics; 37, Broad street buildings, City. C. 1858. *Trans.* 4.

1857 Felix William Lyon, M.D., Lyndhurst, Hants.

1846 William M‘Ewen, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1823 †George Macilwain, Consulting Surgeon to the Finsbury Dispensary and the St. Ann's Society's Schools; 3, the Court yard, Albany. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 Richard Macintosh, M.D.

1859 *John M‘Intyre, M.D.; Odiham, Hants.

Elected

1848 Frederick William Mackenzie, M.D., Senior Physician to the Western General Dispensary; 11, Chester place, Hyde park square. *Trans.* 2.

1818 William Mackenzie, M.D., Surgeon-Oculist to H.M. the Queen in Scotland, and Surgeon to the Glasgow Eye Infirmary; 49, Bath street, Glasgow. *Trans.* 2.

1854 *Draper Mackinder, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1844 Daniel Maclachlan, M.D., Physician to the Royal Hospital, Chelsea, and Deputy Inspector-General of Hospitals; Royal Hospital, Chelsea. C. 1860. *Trans.* 1.

1860 John Maclean, M.D., 24, Portman street, Portman square.

1851 Samuel Maclean, 10, Conduit street, Bond street.

1849 Duncan Maclachlan Maclure, 16, Harley street, Cavendish square.

1842 John Macnaught, M.D., Bedford street, Liverpool.

1837 Andrew Melville M'Whinnie, Assistant-Surgeon to the London Hospital for Diseases of the Skin, Blackfriars; 5, Crescent, New Bridge street, Blackfriars. C. 1851-2. *Trans.* 1.

1855 William Marcet, M.D., F.R.S., Assistant-Physician to, and Lecturer on Chemistry at, the Westminster Hospital; 36, Chapel street, Belgrave square. *Trans.* 1.

1848 William Orlando Markham, M.D., Physician to, and Lecturer on Physiology and General and Morbid Anatomy at, St. Mary's Hospital; 33, Clarges street, Piccadilly. *Trans.* 1.

1824 Sir Henry Marsh, Bart., M.D., Physician to H.M. the Queen in Ireland, Consulting Physician to the City of Dublin Hospital, and Physician to Steevens's Hospital; 9, Merrion square north, Dublin.

1838 Thomas Parr Marsh, M.D., Consulting Physician to the Salop Infirmary, Shrewsbury; Powyn, Merionethshire.

1851 John Marshall, F.R.S., Surgeon to University College Hospital; 10, Savile row, Burlington gardens. *Trans.* 2.

1841 Sir James Ranald Martin, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council; 71a, Grosvenor street. C. 1853.

Elected

1849 George Bellasis Masfen, Ghazeepore, India.

1853 William Edward Masfen, Surgeon to the Staffordshire General Infirmary; Stafford.

1818 J. P. Maunoir, Professor of Surgery at Geneva. *Trans.* 4.

1837 Thomas Mayo, M.D., F.R.S., President of the Royal College of Physicians, and Honorary Physician to the St. Marylebone Infirmary; 56, Wimpole street, Cavendish square. S. 1841. C. 1847-8. V.P. 1851-2.

1839 Richard Henry Meade, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1837 Samuel William John Merriman, M.D., Physician to the Royal Infirmary for Children and Women, Consulting Physician-Accoucheur to the Westminster General Dispensary, and Physician-Accoucheur to the Western General Dispensary; 3, Charles street, Westbourne terrace. C. 1851-2. *Trans.* 1.

1852 James Merryweather, 57, Brook street, Grosvenor square.

1847 Edward Meryon, M.D., *Librarian;* 14, Clarges street, Piccadilly. L. 1859. *Trans.* 1.

1815 Augustus Meyer, M.D., St. Petersburgh.

1840 Richard Middlemore, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.

1854 Edward Archibald Middleship, late of Richmond, Surrey.

1818 *Patrick Miller, M.D., F.R.S.E., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.

1844 Nathaniel Montefiore, 36, Hyde park gardens.

1848 Charles Hewitt Moore, *Secretary*, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 102, Piccadilly. L. 1858. S. 1859. *Trans.* 3.

1836 George Moore, M.D., Hastings, Sussex.

1857 John Morgan, 3, Sussex place, Hyde park gardens.

1854 George Moseley (late of Sandgate).

1851 Frederick John Mouat, M.D., Professor of Medicine in the Medical College of Calcutta, Secretary of the Council of Education in India, and Inspector-General of Gaols, Lower Provinces; Calcutta.

Elected

1856 Charles Murchison, M.D., Assistant-Physician to the London Fever Hospital, and Lecturer on Pathological Anatomy at the Middlesex Hospital; 79, Wimpole street, Cavendish square. *Trans.* 2.

1847 Simon Murchison, Bicester, Oxon.

1859 George Nayler, Assistant-Surgeon to the Warley Depôt; Warley Barracks, Brentwood, and Junior United Service Club.

1835 Thomas Andrew Nelson, M.D., 10, Nottingham terrace, York gate, Regent's park [54, Marylebone road].

1843 Edward Newton, 30, Fitzroy square.

1851 James Nichols, 13, Savile row, Burlington gardens.

1819 *George Norman, Consulting Surgeon to the Bath United Hospital, and Surgeon to the Puerperal Charity; Circus, Bath. *Trans.* 3.

1849 Henry Burford Norman, Portland Lodge, Southsea, Hants.

1845 Henry Norris, Charmouth, Dorset.

1847 *William Edward Charles Nourse, 11, Old Steine, Brighton.

1849 *Arthur Noverre, 25, South street, Park lane.

1859 *Thomas Nunneley, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 1.

1847 Thomas O'Connor, March, Cambridgeshire.

1843 William O'Connor, M.D., Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1846 Francis Odling, 52, Devonshire street, Portland place.

1858 William Mackay Ogilvie, Boughton Blean, near Faversham, Kent.

1858 John William Ogle, M.D., Assistant-Physician to St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 3.

1855 William Ogle, M.A., M.D., 3, Stewart terrace, Derby.

1850 Henry Oldham, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital, and Obstetric Physician to the Tower Hamlets Dispensary; 26, Finsbury square. *Trans.* 1.

Elected

1846 *Edward Latham Ormerod, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.

1847 *William Bousfield Page, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.

1840 James Paget, F.R.S., Surgeon Extraordinary to H.M. the Queen; Assistant-Surgeon to St. Bartholomew's Hospital; 1, Harewood place, Hanover square. C. 1848-9. *Trans.* 8.

1858 *William Paley, M.D., Physician to the Halifax Infirmary; Carlton place, Halifax, Yorkshire.

1836 S. W. Langston Parker, Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.

1847 Nicholas Parker, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 22, Finsbury square.

1841 John Parkin, M.D., Rome.

1851 James Part, M.D., 7, Camden road villas, Camden town.

1828 †Richard Partridge, F.R.S., Professor of Anatomy to the Royal Academy of Arts, Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6. C. 1837-8. V.P. 1847-8.

1845 Thomas Bevill Peacock, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria park; 20, Finsbury circus. S. 1855-6. *Trans.* 2.

1856 Richard King Peirce, 16, Norland place, Notting hill.

1830 Charles P. Pelechin, M.D., St. Petersburgh.

1855 *Oliver Pemberton, Surgeon to the Birmingham General Hospital, and Lecturer on Surgical Pathology at Sydenham College; 18, Temple row, Birmingham.

1844 William Vesalius Pettigrew, M.D., Surgeon to the Female Orphan Asylum, Lambeth; 7, Chester street, Grosvenor place.

1848 Edward Phillips, M.D., F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

Elected

1852 RICHARD PHILLIPS, Winchester place, Claremont square, Pentonville [68, Pentonville road].

1854 THOMAS BACON PHILLIPS, M.D., Physician to the Convalescent Home; 36, Lansdowne place, Brighton.

1846 FRANCIS RICHARD PHILP, M.D.

1851 *JAMES HOLLINS PICKFORD, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1851 JOHN PICTON, M.D.

1836 ISAAC PIDDUCK, M.D., Physician to the Bloomsbury Dispensary; 22, Montague street, Russell square. *Pro.* 2.

1852 HENRY PILLEAU, Staff Surgeon, 1st Class; 21, Kensington square. (India.)

1841 HENRY ALFRED PITMAN, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Consulting Physician to the Royal General Dispensary, St. Pancras; 94, Gloucester place, Portman square. L. 1851-3.

1850 ALFRED POLAND, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital, and Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Bolton row, May fair.

1845 GEORGE DAVID POLLOCK, F.L.S., *Librarian*, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 27, Grosvenor street, C. 1856-7. L. 1859. *Trans.* 2.

1843 CHARLES POPE, M.D., F.L.S., Glastonbury, Somersetshire.

1846 JEPHSON POTTER, M.D., F.L.S., Sussex House, Pittville, Cheltenham.

1842 JAMES POWELL, M.B.

1851 ROBERT FRANCIS POWER, M.D., 7, Lower Grosvenor place.

1857 WILLIAM OVEREND PRIESTLEY, M.D., Lecturer on Midwifery at the Middlesex Hospital, Physician-Accoucheur to the St. Marylebone Infirmary, and Physician to the Samaritan Free Hospital; 31, Somerset street, Portman sq.

1839 JOHN PROPERT, Consulting Surgeon to the Society of Ancient Britons; 6, New Cavendish street, Portland place.

1845 JOHN PYLE, 56, Oxford terrace, Hyde park. C. 1860.

Elected

1816 Sir William Pym, M.D., K.C.H., Inspector-General of Army Hospitals, and Superintendent-General of Quarantine in the United Kingdom.

1830 Jones Quain, M.D., Paris.

1850 Richard Quain, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 23, Harley street, Cavendish square. *Trans.* 1.

1835 Richard Quain, F.R.S., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 Charles Bland Radcliffe, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; 4, Henrietta street, Cavendish square.

1857 Henry Ranke, M.D. (Germany).

1854 William Henry Ransom, M.D., Physician to the Nottingham General Hospital; Nottingham.

1859 Henry Hunter Raymond, 21, Ridgway place, Wimbledon, Surrey.

1858 Frederick George Reed, M.D., 46, Hertford street, May fair.

1821 Henry Reeder, M.D.

1857 George Owen Rees, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Materia Medica at the University of London; 26, Albemarle street, Piccadilly. *Trans.* 1.

1855 John Russell Reynolds, M.D., Assistant-Physician to the Westminster Hospital; 38, Grosvenor street.

1847 Samuel Richards, M.D., 36, Bedford square.

1852 Christopher Thomas Richardson, M.B., Physician to the Metropolitan Free Hospital; 16, Hinde street, Manchester square.

1829 Sir John Richardson, Knt., M.D., LL.D., C.B., F.R.S., Inspector of Hospitals and Fleets; Lancrigg, Grasmere, Westmoreland.

Elected

1849 *William Richardson, M.D., 9, Ephraim road, Tunbridge Wells, Kent.

1845 Benjamin Ridge, M.D., 21, Bruton street, Berkeley square.

1843 Joseph Ridge, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1852 Charles Ridley, Surgeon to the Royal Society for Protection of Life from Fire; 6, Charlotte street, Bedford sq.

1852 John Roberts, M.R.C.P., 75, Grosvenor street.

1829 *Archibald Robertson, M.D., F.R.S., Hon. Physician to the Northampton General Infirmary, Northampton.

1855 Charles Alexander Lockhart Robertson, M.D., Medical Superintendent of the Sussex County Lunatic Asylum, and Hon. Secretary to the Association of Medical Officers of Asylums and Hospitals for the Insane; Hayward's Heath, Sussex.

1857 John George Robertson, Assistant Medical Officer, County Lunatic Asylum; Exminster, Devonshire.

1843 George Robinson, M.D., Physician to the Newcastle-upon-Tyne Dispensary; Eldon square, Newcastle-upon-Tyne. *Trans.* 2.

1843 William Roden, M.D., F.L.S., the Grange, Kidderminster, Worcestershire.

1835 George Hamilton Roe, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 57, Park street, Grosvenor square. C. 1841-2. *Trans.* 1.

1836 Arnold Rogers, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover square.

1846 William Richard Rogers, M.D., Physician to the Farringdon General Dispensary and Lying-in Charity, and to the Western General Lying-in Institution; 56, Berners street.

1819 Henry Shuckburgh Roots, M.D., 2, Russell square. C. 1833, 1845. V.P., 1834-5. *Trans.* 1.

1829 William Sudlow Roots, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 George Roper, 180, Shoreditch.

1836 Richard Roscoe, M.D.

1855 Thomas Tattersall Roscow, M.D.

Elected	
1836	*Caleb Burrell Rose, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.
1857	Henry Cooper Rose, M.D., High street, Hampstead.
1849	Charles Henry Felix Routh, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.
1845	Henry Mortimer Rowdon, M.D., Member of the Court of Examiners of the Society of Apothecaries; 29, Nottingham place, Marylebone road.
1834	Henry Wyldbore Rumsey, Gloucester lodge, Cheltenham.
1845	James Russell, M.D., Physician to the Birmingham General Hospital, and Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.
1851	Henry Hyde Salter, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague street, Russell square.
1856	Samuel James A. Salter, F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 1.
1849	Hugh James Sanderson, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.
1855	John Burdon Sanderson, M.D., Assistant-Physician to the Hospital for Consumption; Medical Officer of Health for Paddington; Lecturer on Medical Jurisprudence at St. Mary's Hospital; 9, Gloucester place, Hyde park.
1847	William Henry Octavius Sankey, M.D., Middlesex County Lunatic Asylum, Hanwell.
1845	Edwin Saunders, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince Consort; 13a, George street, Hanover square.
1834	Ludwig V. Sauvan, M.D., Warsaw.
1859	William Scovell Savory, F.R.S., Lecturer on General Anatomy and Physiology at St. Bartholomew's Hospital, Surgeon to the Great Northern Hospital, and Professor of Comparative Anatomy and Physiology at the Royal College of Surgeons; 13, Charterhouse square. *Trans.* 2.

Elected

1840 Augustin Sayer, M.D., Consulting Physician to the Lock Hospital; 28, Upper Seymour street, Portman square.

1853 Maurice Schulhof, M.D., Physician to the Royal General Dispensary, Bartholomew close; Senior Physician to the Blenheim Free Dispensary; 14, Brook street, Grosvenor square.

1858 *George Scratchley, M.D., New Orleans, Louisiana, U.S.

1856 Edwin Sercombe, Surgeon-Dentist to St. Mary's Hospital; 49, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1824 †Edward James Seymour, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought;' 13, Charles street, Berkeley square. C. 1826, 1831. S. 1827-8. V.P. 1830, 1842. *Trans.* 2.

1840 William Sharp, M.D., F.R.S., Horton house, Rugby. *Trans.* 1.

1837 William Sharpey, M.D., F.R.S., LL.D., Examiner in Anatomy at the University of London, Professor of Anatomy and Physiology in University College, London, and Secretary of the Royal Society; 33, Woburn place, Russell square. C. 1848-9.

1836 Alexander Shaw, *Treasurer*, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 22A, Cavendish square. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-9. *Trans.* 4.

1848 *Edward James Shearman, M.D., Rotherham, Yorkshire.

1859 Septimus William Sibley, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 2.

1849 Francis Sibson, M.D., F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 40, Brook street, Grosvenor square. *Trans.* 1.

1848 Edward Henry Sieveking, M.D., Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester square. C. 1859-60. *Trans.* 1.

1839 Thomas Hookham Silvester, M.D., Medical Officer to the Clapham General Dispensary; High street, Clapham. C. 1854-5. *Trans.* 1.

Elected

1842 JOHN SIMON, F.R.S., Surgeon to, and Lecturer on Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 44, Cumberland street, Bryanston square. C. 1854-55. *Trans.* 1.

1857 JAMES LEWIS SIORDET, M.B., late Physician to the Blenheim Free Dispensary and Infirmary.

1827 GEORGE ROBERT SKENE, Bedford.

1824 †FREDERIC CARPENTER SKEY, F.R.S., *President*, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 13, Grosvenor street. C. 1828. L. 1829-31. V.P. 1841-2. P. 1859. *Trans.* 1.

1852 CHARLES CASE SMITH, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1854 EDWARD SMITH, M.D., LL.B., F.R.S., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest; 16, Queen Anne street, Cavendish square. *Trans.* 5.

1835 JOHN GREGORY SMITH, Harewood, Leeds, Yorkshire.

1843 ROBERT WILLIAM SMITH, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.

1838 SPENCER SMITH, *Vice-President*, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 48, Sussex gardens, Hyde park. C. 1854. S. 1855-8. V.P. 1859.

1845 WILLIAM SMITH, Chesterfield, Derbyshire.

1847 WILLIAM SMITH, M.D., Consulting Physician to the Weymouth Infirmary, Weymouth, Dorsetshire.

1850 WILLIAM TYLER SMITH, M.D., Examiner in Midwifery at the University of London; Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 7, Upper Grosvenor street. *Trans.* 2.

1851 JOHN SODEN, Surgeon to the Bath United Hospital, and Consulting Surgeon to the Bath Eye Infirmary; 24, Circus, Bath. *Trans.* 2.

1816 *JOHN SMITH SODEN, 101, Sidney place, Bath. *Trans.* 1.

Elected

1830 Samuel Solly, F.R.S., Surgeon to St. Thomas's Hospital, and Consulting Surgeon to the Royal General Dispensary, Bartholomew close; 18, St. Helen's place, Bishopsgate street. L. 1838-40. C. 1845-6. V.P. 1849-50. *Trans.* 6.

1844 Frederick Robert Spackman, M.D., Harpenden, St. Alban's.

1834 James Spark, Italy.

1851 Robert John Spitta, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. *Trans.* 1.

1843 *Stephen Spranger, Hursley, Hampshire.

1858 Joshua Harrison Stallard, M.B., Lond., Physician to the St. George's and St. James's Dispensary; 12, Welbeck street, Cavendish square.

1815 †Edward Stanley, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon to St. Bartholomew's Hospital; 23a, Brook street, Grosvenor square. C. 1821-2, 1835, 1845-6, 1852-3. S. 1824. V.P. 1827, 1839-40. T. 1832-4. P. 1843-4. *Trans.* 12.

1857 John Stanton, M.D., 7, Upper George street, Bryanston square.

1851 James Startin, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1858 Edward Stephens, M.D., Consulting Surgeon to the Manchester Lying-in Hospital; 58, Bridge street, Manchester.

1854 Henry Stevens, M.B., late Resident Medical Officer, St. Luke's Hospital for Lunatics, Old street.

1842 Alexander Patrick Stewart, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 74, Grosvenor street. C. 1856-7.

1859 William Edward Stewart, 12, Weymouth street, Portland place.

1856 Alonzo Henry Stocker, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.

1843 Robert Reeve Storks, Paris.

Elected

1858 †John Fremlyn Streatfeild, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 15, Upper Brook street, Grosvenor square.

1839 Alexander John Sutherland, M.D., F.R.S., Consulting Physician to St. Luke's Hospital for Lunatics, and Consulting Physician to the Royal South London Dispensary; 6, Richmond terrace, Whitehall. C. 1850-1. V.P. 1858-9. *Trans.* 1.

1860 Sigismund Sutro, M.D., Senior Physician to the German Hospital; 37a, Finsbury square.

1855 John Maule Sutton, M.D., Kent House, Tenby, South Wales.

1842 James Syme, F.R.S.E., Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland street, Edinburgh. *Trans.* 4.

1854 *Frederick Symonds, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont street, Oxford.

1844 Richard William Tamplin, Surgeon to the Royal Orthopædic Hospital; 33, Old Burlington street.

1848 Thomas Hawkes Tanner, M.D., F.L.S., Assistant Physician for the Diseases of Women and Children to King's College Hospital; 10, Charlotte street, Bedford square.

1852 Robert Taylor, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill street; 10, George street, Hanover square.

1845 Thomas Taylor, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere street, Cavendish square.

1856 Thomas Pridgin Teale, F.L.S., Surgeon to the Leeds General Infirmary; Albion street, Leeds. *Trans.* 2.

1859 Edward Tegart, Junior, 49, Jermyn street, St. James's.

1845 *Evan Thomas, York street, Cheetham hill road, Manchester.

1857 Henry Thompson, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 18, Harley street, Cavendish square.

Elected

1852 HENRY THOMPSON, Consulting Surgeon to the St. Marylebone Infirmary, and Assistant-Surgeon to University College Hospital; 16, Wimpole street, Cavendish square. *Trans.* 2.

1819 †JOHN THOMSON, M.D., F.L.S., Senior Physician to the Finsbury Dispensary; 18, Dalby terrace, Islington, [364, City road]. C. 1833. L. 1834-7. V.P. 1850-1.

1850 ROBERT DUNDAS THOMSON, M.D., F.R.S., Physician to the Scottish Hospital, Examiner in Chemistry at the University of London, and Medical Officer of Health for St. Marylebone; 41, York terrace, Regent's park. *Trans.* 2.

1836 JOHN THURNAM, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.

1848 EDWARD JOHN TILT, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1828 JAMES TORRIE, M.D., Old Bridge of Don, by Aberdeen.

1843 JOSEPH TOYNBEE, F.R.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's Dispensary; 18, Savile row, Burlington gardens. *Trans.* 7. *Pro.* 1.

1850 SAMUEL JOHN TRACY, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington street.

1859 EDWIN THOMAS TRUMAN, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1855 JAMES STEWART TULLOCH, M.D., 1, Pembridge place, Bayswater.

1845 THOMAS TURNER, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine; 77, Mosley street, Manchester.

1846 ALEXANDER URE, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour street, Portman square. *Trans.* 1.

1806 BOYER VAUX, M.D.

Elected

1810 James Vose. *Trans.* 1.

1828 Benedetto Vulpes, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1854 Edward Waddington, Surgeon to the King's Own Staffordshire Rifles; Wakefield, Yorkshire.

1841 Robert Wade, Senior Surgeon to the Westminster General Dispensary; 68, Dean street, Soho. *Trans.* 1.

1852 Walter Hayle Walshe, M.D., Professor of the Theory and Practice of Medicine in University College, London, and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. *Trans.* 1.

1851 Henry Haynes Walton, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital; 69, Brook street, Hanover square. *Trans.* 1. *Pro.* 1.

1852 Daniel Wane, M.D., Obstetric Physician to the Blenheim Free Dispensary; 20, Grafton street, Berkeley square.

1846 Nathaniel Ward, Assistant-Surgeon to the London Hospital; 1, Broad-street buildings, City. C. 1857. *Trans.* 1.

1821 William Tilleard Ward, Duncannon House, Brighton.

1858 John Richard Wardell, M.D., 4, Belmont, Tunbridge Wells.

1846 James Thomas Ware, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 51, Russell square.

1818 John Ware, Clifton, near Bristol.

1814 †Martin Ware, 51, Russell square. C. 1844-5. T. 1846. V.P. 1853.

1829 Elias Taylor Warry, M.D., Yeovil, Somerset.

1837 Thomas Watson, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1847 *Thomas Watson, L.R.C.P. Edinb., Dolby House, Holbeach, Lincolnshire.

1854 William Webb, M.D., Wirksworth, Derbyshire.

Elected

1840 William Woodham Webb, M.D., Cliff House, Kirtley, South Lowestoft, Suffolk.

1842 Frederic Weber, M.D., Assistant-Physician to the Middlesex Hospital; 44, Green street, Park lane. C. 1857.

1857 Hermann Weber, M.D., Physician to the German Hospital; 49, Finsbury square. *Trans.* 1.

1835 †John Webster, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 24, Brook street, Grosvenor square. C. 1843-4. V.P. 1855-6. *Trans.* 6.

1844 William Wegg, M.D., Physician to the St. George's and St. James's Dispensary; 49, Maddox street, Hanover square. L. 1854-8.

1854 Thomas Spencer Wells, Lecturer on Surgery at the Grosvenor-place School of Anatomy and Medicine, and Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 1. *Pro.* 1.

1816 Sir Augustus West, Knt., M.D., Deputy-Inspector of Army Hospitals to the Portuguese Forces; Paris.

1842 Charles West, M.D., Examiner in Midwifery at the University of London; Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. *Trans.* 2.

1841 Thomas West, M.D., Daventry, Northamptonshire.

1828 John Whatley, M.D.

1849 John White.

1852 John Wiblin, Medical Inspector of Emigrants and Recruits; 73, Morland place, Southampton.

1824 *William John Wickham, Consulting Surgeon to the Hants County Hospital; Winchester, Hants. *Trans.* 1.

1844 Frederic Wildbore, 1, Trafalgar place east, Hackney road.

1837 George Augustus Frederick Wilks, M.D.

1860 Arthur Wynn Williams, M.D., 20, King street, Portman square.

Elected

1840 Charles James Blasius Williams, M.D., F.R.S., *Vice-President*, Consulting Physician to the Hospital for Consumption; 49, Upper Brook street, Grosvenor square. C. 1849-50.

1859 *Charles Williams, House-Surgeon to the Norfolk and Norwich Hospital; Norwich.

1859 Joseph Williams, M.D., 8, Tavistock square.

1829 Robert Willis, M.D., Barnes, Surrey. L. 1838-41.

1839 †Erasmus Wilson, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.

1839 James Arthur Wilson, M.D., 28, Dover street, Piccadilly. C. 1846-7. V.P. 1858-9. *Trans.* 4.

1850 *Robert Stanton Wise, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 Thomas Alexander Wise, M.D., F.R.S.E., Rostellan Castle, Rostellan, County Cork.

1841 George Leighton Wood, Surgeon to the Bath General Hospital; 27, Queen square, Bath.

1851 John Wood, Assistant-Surgeon to King's College Hospital; 4, Montague street, Russell square. *Trans.* 1.

1848 William Wood, M.D., 54, Upper Harley street.

1843 John Ward Woodfall, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.

1833 †Thomas Wormald, Assistant-Surgeon to St. Bartholomew's Hospital, and Surgeon to the Foundling Hospital; 42, Bedford row. C. 1839. V.P. 1854.

1842 William Collins Worthington, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1848 Edward John Wright, 13, Montague place, Clapham road.

1855 Henry G. Wright, M.D., Physician to the Samaritan Free Hospital for Women and Children, and to the St. Pancras Royal General Dispensary; 23, Somerset street, Portman square.

[It is particularly requested, that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected	
1841	William Thomas Brande, D.C.L., F.R.S., Hon. Professor of Chemistry at the Royal Institution of Great Britain, Examiner in Chemistry, and Member of the Senate of the University of London ; Royal Mint, Tower hill.
1835	Sir David Brewster, K.H., D.C.L., LL.D., F.R.S., &c., Corresp. Memb. Institute of France, Principal and Vice-Chancellor of the University of Edinburgh.
1853	Benjamin Collins Brodie, M.A., F.R.S., Aldrichian Professor of Chemistry in the University of Oxford.
1847	Edwin Chadwick, late Commissioner of the Board of Health.
1835	Michael Faraday, D.C.L., F.R.S., Corresp. Memb. Institute of France, Member of the Senate of the University of London, and Fullerian Professor of Chemistry in the Royal Institution.
1857	William Farr, M.D., D.C.L., F.R.S., General Register Office, Somerset House.
1841	Sir John Frederick William Herschel, Bart., D.C.L., F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.
1835	Sir William Jackson Hooker, K.H., D.C.L., LL.D., F.R.S., F.L.S., Director of the Royal Botanic Gardens, Kew ; West Park, Kew.
1847	Richard Owen, D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France (Foreign Associate of the Academy of Sciences) ; Superintendent of the Natural History Departments in the British Museum ; Sheen Lodge, Mortlake.
1859	John Thomas Quekett, F.R.S., F.L.S., Conservator of the Museum, and Professor of Histology at the Royal College of Surgeons of England.
1825	The Rev. Adam Sedgwick, A.M., D.C.L., F.R.S., Woodwardian Professor of Geology, Cambridge.
1841	The Rev. William Whewell, D.D., F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected	
1841	G. Andral, M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French, Professor of Pathology in the Faculty of Medicine; Paris.
1856	Baron Paul Dubois, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, Dean of, and Professor of Clinical Midwifery in, the Faculty of Medicine; Paris.
1835	Carl Johan Ekströmer, M.D., C.M., K.P.S., and W., Physician to the King of Sweden, President of the College of Health, and Director-General of Hospitals; Stockholm.
1841	Christian Gottfried Ehrenberg, Member of the Institute of France; Berlin.
1859	J. Henle, M.D., Professor of Anatomy at Göttingen.
1841	James Jackson, M.D., LL.D., Emeritus Professor of Medicine in the University of Cambridge, Boston, U.S.
1856	Bernhard Langenbeck, M.D., Professor of Surgery in the University of Berlin.
1843	Baron Justus von Liebig, M.D., Conservator of the Royal Collection, and Professor of Chemistry in the University of Munich.
1841	P. C. A. Louis, M.D., Honorary Physician to the Hôtel-Dieu, Member of the Imperial Academy of Medicine; Paris.
1847	Carlo Matteucci, Professor in the University of Pisa, Member of the Institute of France.
1853	Valentine Mott, M.D., LL.D., Emeritus Professor of Surgery in the University of New York, late President of the New York Academy of Medicine; New York.
1841	Bartolomeo Panizza, M.D., Pavia.

Elected	
1859	Pierre Rayer, M.D., late Physician to the "Hôpital de la Charité," Commander of the Legion of Honour, Member of the Institute, and of the Imperial Academy of Medicine; Paris.
1850	Carl Rokitansky, M.D., Curator of the Imperial Pathological Museum, and Professor at the University of Vienna.
1856	Louis Stromeyer, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.
1835	Friedrich Tiedemann, M.D., Frankfort-on-the-Maine.
1856	A. Velpeau, Member of the Institute, and of the Imperial Academy of Medicine, Professor in the Faculty of Medicine, Surgeon to the "Hôpital de la Charité;" Paris.
1856	Rudolph Virchow, M.D., Professor of Pathological Anatomy in the University of Berlin.
1859	W. Vrolik, M.D., Professor of Natural History at Amsterdam.

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HISTORY OF AN ADDITIONAL CASE
OF
TUBAL GESTATION.

[SEE VOL. XLI, PAGE 137.]

BY

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Received Oct. 18th.—Read Nov. 8th, 1859.

On Monday, the 10th of October, 1859, Mr. John Gregory Forbes related to me the case of a woman who had died suddenly the previous day from internal hæmorrhage. The catamenia had appeared one month before her death, and had gone on properly, with the intermission of one week. On Saturday morning, the 8th of October, she was seized with pain in the uterus, and sense of bearing down. Mr. Forbes saw her in the evening at half-past eight o'clock, when the extremities were cold, the pulse feeble, the complexion bloodless, and she was complaining of some pain in the lower part of the abdomen, which was tender on pressure. She had taken no food since the night before, and had drunk only some brandy and water. There was no suspicion of pregnancy, as she had been unwell only a few days before. Warm fomentations were applied to the abdomen, and brandy and water administered. She was able to speak, so that there were hopes of her recovery. But early on Sunday morning, soon after sitting up, and taking one drachm of castor-oil, which had been recommended, she lay down, and died in a short time, having spoken to her mother before about her children.

Mr. Forbes examined the body the following day, and found three pints of blood in the sac of the peritoneum, which had escaped from an opening in the left Fallopian tube, which contained an ovum. The uterus was laid open and examined, and Mr. Forbes has informed me that no trace of decidua could be discovered lining the cavity. The uterus was not allowed to be taken away, but the Fallopian tube and ovarium were permitted to be removed, and very kindly presented to me for minute anatomical examination, and are now placed on the table of the Royal Medical and Chirurgical Society.

A small corpus luteum, perfectly formed, is seen in the ovarium. The Fallopian tube, to a great extent, had been laid open before it was presented to me; and Mr. Forbes has informed me, that when this was done, the embryo was not seen, nor the amnion nor vesicula umbilicalis. On placing the preparation under alcohol, and examining it, I found a deciduous membrane adhering to the inner surface of the tube, and enclosing the villi and membrane of the chorion everywhere, as in all the preparations of tubal gestation described in the forty-first volume of the 'Medico-Chirurgical Transactions.' The cells or interstices of the villi of the chorion are now seen partially filled with coagulated blood. The coats of the tube at the outer extremity of the dilated portion, are extremely thin; there is seen an oblong aperture, with a thin, not very irregular margin, in the posterior part of the coats of the tube, and an opening of the same form and extent in the decidua, which had adhered to this part of the tube. A slender coagulum of blood is still seen hanging out of these openings. There is no appearance of any unusual vascularity in the coats of the tube around the opening, and in the recent state there was no appearance of any artery or vein having ramified around this aperture, from which the three pints of blood found in the sac of the peritoneum could have escaped.

By the openings formed in the coats of the tube and corresponding part of the membrana decidua, a communication was established between the cells of the villi of the chorion and placenta and the sac of the peritoneum, and through these apertures the blood must have flowed from the cells of the chorion into the abdominal cavity, until the patient died; and it must be obvious that nature had here provided no means of arresting the flow of blood, as in cases of uterine hæmorrhage; and that in all similar cases there must be a fatal result.

In all perfect human ova, whether found in the Fallopian tube, or in the cavity of the uterus, or expelled from the uterus in abortion, the cells of the placenta and chorion are found distended with fluid or coagulated blood. This maternal blood is first conveyed by the arteries of the placental decidua, from the arteries of the uterus, into the cells of the placenta, from thence it flows into the cells of the villi of the chorion, from whence it is conveyed into the decidual cavity by the veins of the decidua reflexa, and by the veins of the uterine decidua, or decidua vera, into the veins of the uterus. If a communication be formed between the cells of the placenta or chorion and the abdominal cavity, the blood will flow from these cells, through the opening formed, into the sac of the peritoneum, and the patient will inevitably perish.[1]

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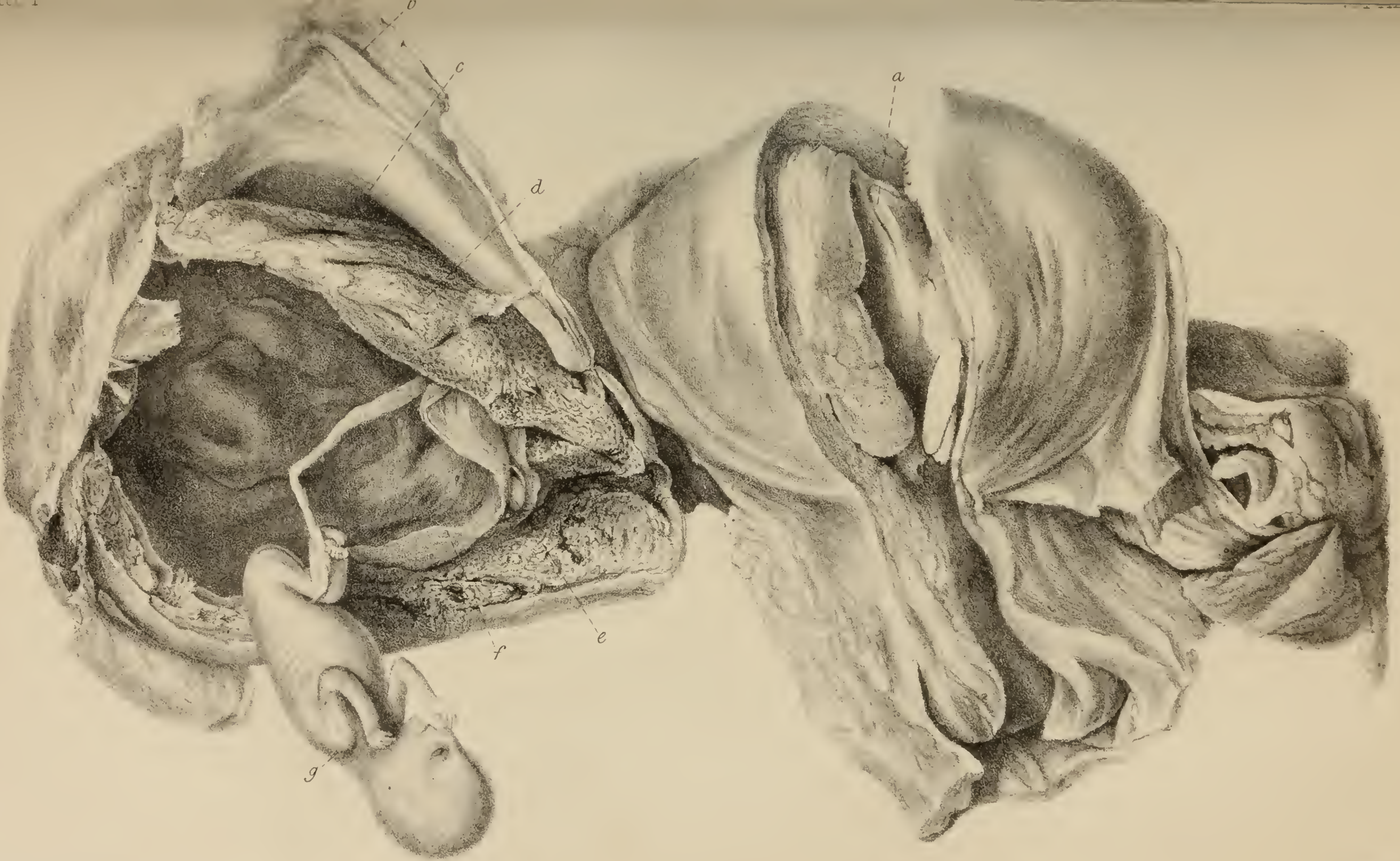
[1] See a paper "On the Circulation of the Maternal Blood in the Human Ovum during the Early Months." ('Medical Gazette,' 1840, vol. xxvi.)

EXPLANATION OF PLATE I.

The drawing is a representation of the uterus and right Fallopian tube, containing a perfect ovum; described in the 'Medico-Chirurgical Transactions,' vol. xli, page 141.

a. A thick layer of coagulated fibrine, in which no blood-vessels could be discovered, lining the uterus.
b. The lining membrane of the Fallopian tube.
c. The decidua which surrounds the entire ovum, and which adhered to the inner membrane of the tube, corresponding with the uterine decidua in ordinary pregnancy.
d. The interstices of the villi of the chorion, filled partially with coagula of maternal blood.
e. The vesicula umbilicalis between the chorion and amnion, near the placenta, the slender peduncle of which is seen in the preparation proceeding to the umbilical cord.
f. The amnion.
g. The embryo perfectly formed.



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W West imp

ON

THE ETIOLOGY AND TREATMENT

OF

PERITONITIS.

BY

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ASSISTANT-PHYSICIAN TO, AND LECTURER ON MATERIA MEDICA AND THERAPEUTICS AT, GUY'S HOSPITAL.

Received Dec. 13th.—Read Dec. 13th, 1859.

The knowledge of the causes of disease is in many instances the best guide to treatment; and the value of pathological records is especially shown in affording safe indications for therapeutical measures; for if we can discover and remove disturbing forces, the functions of life will gradually re-assume their wonted state. Eminent writers on medical science have dwelt upon the importance of regarding local disease as arising from a general or constitutional change in the system; and whilst all acknowledge that fact, and many bear it continually in view, still, some diseases are considered as idiopathic, which further investigation proves to have a constitutional origin.

This remark applies with great force to *peritonitis*, for although spoken of and treated as an idiopathic disease, we do not find that it has a local character, unless excited by injury to the serous membrane, or by the direct propagation of disease; and in this aspect, the facts educed from the records of a large number of examinations after death have much interest. In 3752 inspections recorded at Guy's Hospital during a period of twenty-five years, 500 instances of peritonitis occur; but we cannot find a single case, thoroughly detailed, where the disease could be correctly regarded as existing solely in the peritoneal serous membrane.

In relation to the causes of peritonitis, the cases just referred to may be divided into three classes.

1. Peritonitis produced by the extension of disease from adjoining viscera, or excited by direct injury, including cases of perforation of viscera, extravasation, violence, &c.

2. Peritonitis connected with blood-changes, as where inflammation of the serous membrane occurs in the course of albuminuria, pyæmia, puerperal fever, erysipelas, &c.

3. Peritonitis caused by general nutritive changes of the system, which have been followed by acute or chronic disease of the peritoneum, such as struma, cancer, &c.; and comprising also those cases in which the circulation of the peritoneum has been so altered by continued hyperæmia (modifying its state of growth), that very slight exciting causes suffice to induce acute mischief, as occurs in peritonitis with cirrhosis, disease of the heart, &c.

It will be evident that the first class might be regarded as disease of a local, and the second and third as of a general character. The *first* division, by far the largest, includes 261 instances of peritonitis, which was produced in 102 by hernia, internal and external, intussusception, bands of adhesion, and cancerous obstruction. In 19 cases the obstruction was of an internal kind, and in not a few of these death followed from rupture of the intestinal coats. In internal obstruction the intestine above the stricture becomes enormously distended, the mucous membrane is stretched, and presents evidence of great congestion and acute inflammation; the mucous surface is often covered with a diphtheritic layer, numerous small patches of ulceration are found arranged in a linear manner in the direction of greatest tension, and, after a short time, the several coats of the intestine give way, and extravasation takes place.

In the treatment of these cases of internal disease, very different measures have been used, and one almost as unsuccessfully as the other; gastrotomy has been performed, warmth or cold applied, manipulation and changed positions of the patient tried, injections of all kinds administered, and the most powerful depressing agents used; but all now acknowledge that purgatives are exceedingly injurious, and that the most efficacious means of prolonging life is the frequent administration of opium. For by allaying violent peristaltic action, and the spasmodic contraction of the involuntary muscular fibre of the canal, vomiting is often checked, and the perfect rest thus obtained has been followed, as in some cases of cancerous obstruction, by relief to the more urgent symptoms, and by a considerable postponement of the fatal result. Many persons, however, combine calomel with the opium, from the idea that it checks inflammatory action; but if we bear in mind the condition of the mucous membrane above the injury, enormously stretched, acutely inflamed, often ulcerating, and closely resembling a leg of which the skin is stretched to the utmost by anasarcous effusion, followed by a series of minute ulcerative patches, we can scarcely regard mercury as otherwise than injurious; and if perforation take place, as is too often the case, this remedy is not likely to localize the extravasation.

Under the first division we also enumerate 35 cases caused by injuries or operations directly affecting the serous membrane. In some instances of severe abdominal injury, death had resulted before any sign of inflammation had taken place; while in others, the injury had been of such a character, that no treatment could be more than palliative in the most trifling degree, as when the jejunum was completely divided by a vehicle passing over the body: but the museum of Guy's Hospital contains a specimen, where severe injury to the abdomen took place, and death ensued in about three months, and where a small laceration of the liver had evidently become united.

Among the operations referred to, one was for the removal of an ovarian cyst; one a case of gastrotomy; fourteen were cases of paracentesis abdominis, which was performed in five to relieve ascites accompanying cirrhosis, in two that with heart-disease, and in seven to empty large ovarian cysts.

In instances of ascites from heart-disease, chronic bronchitis, and cirrhosis, the whole of the peritoneal capillaries are in a state of continued hyperæmia; the serous membrane becomes opaque and thickened; and a very slight, fresh exciting cause is sufficient to produce acute disease. It is probable, that if a larger number of instances of paracentesis abdominis had been taken, it would have been found, that in ovarian disease, paracentesis is much less frequently followed by severe and fatal result, than in ascites following cirrhosis.

It was not certain that any direct injury had been received in two cases under this class; but great probability favoured such a conclusion. One of these was a young man, Robert P—, æt. 26, who experienced in the night sudden pain in the right side; this continued with varying degrees of severity, and with the ordinary symptoms of peritonitis; death took place in three months, and a collection of pus was found between the liver, diaphragm, and spleen, with general peritonitis. In the second instance referred to, a small abscess was found in a patient, aged sixty-one, at the lower part of the peritoneal cavity, localized, and associated with pneumonia; the disease was possibly pyæmic in its character.

In all these cases of acute peritonitis arising from injuries or operations, absolute rest, in order to promote the localization of the mischief, is one of the great objects to be sought for; and opium serves to effect this purpose as far as any agent we can use; but we may add, that in our experience the union of calomel with the opium appears to *diminish* rather than increase its beneficial influence.

As to the treatment of peritonitis arising from perforation as the result of disease, the same remark applies very forcibly, the adoption of the plan originally proposed by Dr. Stokes and Dr. Graves has been followed by the prolongation of life, and even by recovery.

Of 56 cases of perforation of intestine opening into the peritoneal sac, nine were instances of perforation of the stomach, of which one was caused by cancerous ulceration, one by poisoning with sulphuric acid, and seven were simple ulceration: of these latter, it is interesting to notice the ages of the patients; five were young women, æt. 20, 20, 17, 16, and 21; and two were men, æt. 29 and 37.

Fifteen instances of perforation occurred in the course of continued fever. The fatal lesion took place at the lower part of the ileum, generally about the twenty-first or twenty-second day of the fever, and several perforations were, no doubt, produced, or at least accelerated, by removal to the hospital; in one it was said to be on the tenth day of fever, but some doubt may be attached to this statement, from the difficulty of obtaining an exact history.

In connection with fever as a cause of peritonitis, we may here mention five other cases, in two of which the perforation was not complete, the peritoneum being left; but probably transudation had taken place in a sufficient degree to produce acute mischief; in a third case, the intestine had given way, but local abscess was the result, rather than general acute peritonitis, and life was consequently prolonged; in a fourth chronic adhesions had formed; and in the fifth, described as a case of fever, we find that there were vomicæ in the lungs, and only very slight ulceration in the ileum.

Four cases of perforation of the ileum occurred in connection with strumous disease, leading to sudden and acute peritonitis; one was a child aged five years, with tubercular disease; the second, a female aged thirty-five, a strumous subject, with extensive ulceration of the ileum; the third, a patient with phthisis, aged forty-three; and the fourth, a case of much interest, in which the peritonitis commenced in the pelvic viscera, and the perforation took place, as we find in several cases of fæcal abscess, from the peritoneal to the mucous surface,—it was the case of Ruth S—, æt. 25, in whom, after exposure to cold and wet, menstruation was suddenly checked, and strumous disease of the Fallopian tubes followed, with tubercular deposit in the lungs and peritoneum; local suppuration took place, the intestine was perforated, and fatal peritonitis occurred.

In eleven instances perforation occurred at the cæcum or appendix, nine arising from concretion or ulceration in the latter part.

Five cases of perforation were produced by cancerous disease of other parts, one of the uterus and vagina, and four of the colon; in one of the latter it was also associated with abscess of the liver.

In two cases ovarian disease had led to union with the intestine, perforation, and then extravasation; in one of these the transverse colon was the part perforated.

In ten others, to which we have before referred, hernial obstruction, or intussusception, was followed by rupture of the coats of the bowel.

Of the cases of perforation, then, we find—

10 from hernia,
9 from disease of the stomach,
15 from fever-ulceration of the ileum,
4 from strumous disease,
11 from disease of the cæcum and appendix,
1 from cancer of the vagina,
4 from cancer of the colon,
2 from ovarian adhesions.

56

Under the division of peritonitis from local causes, we next refer to 19 cases where extravasation was so circumscribed as to lead to *fæcal* abscess, and many of these were cases of much interest: thus, Elizabeth H—, æt. 20, was a patient with extra-uterine fœtation, in whom an abscess formed at the lower part of the abdomen, having a large opening into the sigmoid flexure of the colon.

Three arose from ovarian tumours; three from pelvic suppuration—one after labour, a second from a fall, and a third, in a patient aged thirty, in whom, after miscarriage, pelvic inflammation and suppuration took place, and an opening formed into the colon and small intestine. Three were from ulcerated colon in one of which, dysentery, with sloughing, also existed. Five were cases of strumous disease, with tubercular lung or peritoneum; and one of these was a child aged six, in whom the fæcal abscess extended to the umbilicus, from which discharge took place. In one of the cases of fæcal abscess, cancer of the colon existed; in another, disease of the appendix cæci; in a third, the cæcum itself was affected; and in a fourth, a fall produced local disease, ulceration, and extravasation: two of the latter are elsewhere enumerated.

In 42 cases the peritonitis was caused by extension of disease from the bladder, uterus, pelvic viscera, &c., in the following proportion:

6 from ovarian disease,
1 from ulcerated vagina and uterus,
1 from strumous disease of the testicle and castration,
1 from diseased prostate,
1 from strumous pyelitis,
1 from fistula in ano,
1 from cancerous disease of the bladder,
1 from polypus in the bladder,
14 from cystitis, calculus in the bladder, stricture, &c.,
1 from extravasation of urine,
2 from sloughing perinæum,
1 from sloughing of the nates,
10 from lithotomy,
1 from diseased hip and pelvis.

42

With the exception of the first, namely, ovarian disease, few persons would treat these cases of peritonitis from pelvic mischief actively; the removal of the primary source of irritation by every available means, rest, and the mitigation of pain and nervous prostration by narcotics, anodynes, &c., constitute the principal objects of treatment. In the ovarian form of inflammation we find cæcal disease sometimes closely simulated; but even then, local depletion, rest, warmth, opiates, and salines, will, as far as we have observed, effect all that treatment can accomplish beneficially; to give mercurials would, we think, be here uncalled for, and in all the other cases just mentioned it would be absolutely injurious.

In 11 instances, the peritonitis followed disease of the liver and gall-bladder, by simple extension of the disease; in six, abscess of the liver existed; one of these was a child aged two years, the abscess arising after scarlet fever, and local peritonitis was the result. The cause of the suppuration in the liver, in one of the cases, was not at all manifest; it occurred in a dressmaker, aged twenty-eight, without other described disease. A case of more chronic peritonitis arose from hydatids in the liver. In one instance there was rupture of the gall-duct, from impacted calculus; in another, disease set up in the gall-bladder, led to enormous distension; and in a third there was suppuration of the cellular tissue, in connection with biliary calculus.

In the following instance, it is probable that the peritonitis was secondary to disease of the liver, but there is much doubt as to its precise character; we believe it was one of the acute forms of cirrhosis, quickly passing on to suppurative changes, and followed by peritonitis, consequent on the direct extension of disease to the serous membrane.

Acute and chronic peritonitis. Suppuration in the course of the portal veins.

Samuel A—, æt. 35, was admitted into Guy's Hospital February 11th, 1857, and died on the 14th. Nine days before Christmas he had been seized with rigor, pain in the chest, and vomiting, followed by tenderness of the abdomen. He was obliged to give up work; the pain continued in the abdomen, and after a time vomiting of greenish fluid came on. During the whole of the illness he had repeated rigors. He was a spare man, but not emaciated, and slightly jaundiced; the abdomen was full and tender; there was constant vomiting of bilious fluid, and the bowels were confined. The precise character of the disease was obscure till death.

With the exception of pleuritic adhesions, the thoracic viscera were healthy. The peritoneal cavity contained three or four pints of serum, and there was much lymph floating in it; the intestines were matted together, and separated from each other with difficulty. At the upper part of the abdomen, the disease was older, and the adhesions more firm; the colon, stomach, and liver were firmly adherent, and pus was confined in spaces between the viscera; a large quantity of pus was also found between the spleen and the diaphragm. Upon cutting through the lesser omentum, and separating the liver from the duodenum, thus dividing the portal vessels, a large quantity of pus escaped from Glisson's capsule, and from the portal vein. Behind the pancreas was a circumscribed abscess, holding about half a pint of pus. The pancreas was healthy, as was also the duct; so also the splenic and mesenteric veins. In the liver, there was extensive suppuration in connection with the veins; the portal veins were quite distended with pus, their walls softened, and in some parts detached from the tissue around them. In the large veins, distinct flakes of lymph adhered; and in some the lymph was found as a firm stratum lining the cavity of the vessel. The mucous membrane of the intestines was healthy.

Peritonitis is sometimes the result of acute disease of the mucous membrane of the intestine, extending to the deeper coats, and to the peritoneum itself, leading to inflammation, or to perforation and fæcal abscess. Ulceration of the colon was the cause of fæcal abscess in three of the instances we have previously mentioned, and one case of simple perforation, also previously referred to, was from this cause. In three other instances, acute dysentery, with sloughing of the coats of the intestine, also produced peritonitis.

James V—, æt. 30, employed at lead-works, was admitted, with constipation of five days' duration. Powerful medicines were given, and diarrhœa of a dysenteric character came on. The mucous membrane of the colon was found in a sloughing state, and the peritoneum acutely inflamed. Two others were also cases of acute dysentery; the coats of the intestine were most extensively destroyed; in one extravasation was only prevented by adhesions, and in the other the submucous and subperitoneal coats of the intestine contained isolated collections of pus in the form of abscesses, and the mucous membrane of the colon was converted into black, ragged sloughs.

Disease of the cæcum and appendix is another direct cause of peritonitis. Twelve were from this source; nine of these have already been referred to in connection with perforation into the peritoneal cavity; of the three others, the affection of the peritoneum was less severe, and not the immediate cause of death. One was a young person, aged twenty-two, in whom an ovarian cyst burst into the cæcum; no general fæcal extravasation took place, and the fæcal disease might be regarded as secondary.

Another case was that of a boy, aged fourteen, admitted in 1834, with symptoms attributed to fever and bronchitis; a tumour existed in the right hypochondriac region, which was ascribed to disease of the liver. After death, it was found that a local peritoneal abscess existed between the right lobe of the liver and diaphragm; the abscess extended downwards by the side of the ascending colon, and in it the appendix cæci was found perforated. A second opening had also formed into the ascending colon.

The third case was that of Michael R—, aged thirty-four, admitted with diarrhœa, and a painful swelling in the region of the cæcum. The swelling became more prominent in the right iliac fossa, and was opened externally; pus was discharged. The patient gradually sank, and was found to be affected with phthisis, and with fistula in ano. The cæcum was bound down to Poupart's ligament, and the abscess communicated by a sinuous canal with the cæcum close to the valve.

We have thus enumerated the largest division of instances of peritonitis, and we believe in each case it was not due to disease commencing in the peritoneum itself, but that in all the serous membrane became directly affected by the extension of disease to it.

From hernia, of which 19 were internal obstruction .	102
,, injuries or operations . . .	35
,, perforations of the stomach, ileum, cæcum and appendix, colon, &c. (other 13 mentioned with hernia or with cæcal disease) .	43
and leading to fæcal abscess (2 cases otherwise mentioned)	17
,, typhoid ulceration without perforation .	5
,, disease or operations on the bladder and pelvis, viscera, &c.	42
,, disease of liver and gall-bladder, &c. .	11
,, acute disease of the colon (3 others enumerated with perforations) . . .	3
,, diseases of cæcum or appendix (9 others previously mentioned) . . .	3
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In each of these instances, as far as medicinal treatment can be of service, we believe the plan suggested by Dr. Stokes and Dr. Graves, in instances of perforation of the stomach, to be of the greatest value, in promoting the rest of the intestines and the localization of the mischief, the mitigation of suffering, and the alleviation of nervous prostration and collapse, and the facilitation of remedial processes. In many instances, this opiate plan may be combined with local depletion, the external application of anodyne remedies or counter-irritants; but we think that mercury in the form of gray powder or calomel is injurious rather than otherwise, tending to prevent adhesions, by exciting action of the bowels, by increasing the ulceration, and the depression consequent on the disease (which depression is often the *immediate* cause of death), and lastly, by rendering the intestinal contents more fluid, thereby increasing extravasation. We are well aware that many instances of acute peritonitis from diseased cæcum, enteritis, or ovarian disease, recover after mercury has been given; but as far as the cases we have mentioned can be any guide, we are at a loss to know how they would be benefited by mercurial action.

Effervescent medicines generally increase the painful distension of the abdomen; but diaphoretics and salines are sometimes of value, when combined with opium. We do not enter into the subsequent treatment, further than to state, that if the bowels remain inactive, it is far better, in most cases, to use gentle enemata, than to administer aperients by the mouth. The inculcation of rest, and the proper regulation of the diet, are to be borne in mind, as of the greatest moment. When perforation has taken place, scarcely anything should be taken by the mouth; a few teaspoonfuls of fluid, to relieve thirst, will suffice; if large quantities of fluid be swallowed, they will probably pass into the peritoneum, and take away the chance of recovery. Castor-oil has thus been seen, having floated into the serous cavity. In other instances, the most bland forms of nutriment only should be given, and stimulants with great caution, when prostration absolutely calls for them. The return to solid food is often followed by the renewal of acute symptoms, and requires great care.

We now turn to the *second* division, namely, peritonitis connected with a changed condition of blood, such as exists in albuminuria, in pyæmia, in puerperal peritonitis, and in erysipelas, &c. Ninety-four instances of this kind occur out of the total number under consideration.

Sixty-three were cases of *albuminuria;* where the renal disease was of an acute kind, the lymph effused, and the previous symptoms were often well marked; but in some cases, in which there was small granular kidney, with contracted liver, the peritoneum was thickened, and more chronic disease had also existed. Albuminuria is a frequent cause of serous inflammation affecting the pleura, the pericardium, the arachnoid, sometimes the joints, and, as these cases show, the peritoneum also. It rarely, however, happens that the peritoneum alone is affected, although such is sometimes the case. The symptoms also are often masked by the distress arising from the general anasarca, and the dyspnœa from œdematous lungs, &c. The best treatment for these local affections is that adopted for the general disease—diaphoretics, sometimes counter-irritation, or cupping on the loins, free evacuation from the bowels, &c.; but mercurial preparations very readily affect the system, producing severe salivation, without corresponding benefit. One of these cases was connected with purpura, and in another the peritonitis was very severe, accompanied by epilepsy.

Ten of the ninety-four were cases of puerperal peritonitis, and in nearly all suppuration was found, either in the uterine veins, or in the pelvic cellular tissue, or in the broad ligaments. Almost of a similar kind are instances of peritonitis connected with *pyæmia*, following operations or local suppuration. Of these we find thirteen, and five others with erysipelas.

There are sometimes, however, instances in which the peritonitis evidently arises from a general cause, and may, perhaps, be considered pyæmic, or even of a *rheumatic* character, or connected, as we have before said, with renal disease. Three were of this description: one with pericarditis and pleurisy; a second with pneumonia and dysentery; and a third with pericarditis, pleuro-pneumonia, and renal disease discovered after death, there being no albumen in the urine. We subjoin the details of the first and third of these cases.

Hypertrophy of the heart, adherent pericardium, acute pericarditis, pleurisy, and peritonitis.

James M—, æt. 9, was admitted into Guy's, December 12th, 1855. He was a delicate boy, who had suffered from cough, but there was no history of rheumatism; he had been in the hospital for disease of the bones of the foot, and left nearly well; in one week he returned very ill, and was found to be suffering from pericarditis; there was slight pain in the shoulder, but no swelling of the joints generally. He died in three days.

There was a cicatrix on the foot, showing the part from which the fourth metatarsal bone had been removed; but there was no suppuration. There was general pleurisy on both sides, recent lymph in small quantities being found; the lungs were congested, and the bronchi full of tenacious mucus. There were acute and chronic pericarditis, as shown by adhesions of recent lymph, and in some parts by very firm fibrinous bands. Minute vegetations were found on the valves. In the abdomen, although the serous membrane had not lost its transparency, there were some flakes of recent lymph and a small quantity of serum.

Was this general disease of the character of rheumatism? We must regard it as arising from some general cause, and in that respect very different from idiopathic peritonitis.

Acute peritonitis, pericarditis, pleuro-pneumonia. Small, granular kidneys.

William B—, æt. 42, was admitted into Guy's Hospital, June 13th, 1855; he had been a labourer, and had resided in the Borough. A year previously he had had jaundice; and three days before admission had had rigors, but on the following day, although feeling ill, he went to his employment, but the next day he gave up work. On admission he was very ill, presenting the signs of pneumonia of the right lung; the dulness rapidly increased, with bronchophony; much blood was expectorated, and he became delirious, but whilst sensible did not complain of pain. The urine was *not* albuminous. He died on the 20th. There was recent consolidation of the whole of the right lung; the pericardium was covered with a layer of fresh lymph; there was also acute peritonitis, the intestines being adherent by recent lymph. There was a hydatid cyst in the liver, surrounded by a dense white envelope, one eighth of an inch in thickness, and containing disintegrated membrane with opaque fluid; the gland itself was fatty.

The kidneys were granular and coarse, the tunic adherent, the secreting tubes filled with inflammatory products.

In this case, renal disease was followed by acute mischief of serous membranes and pneumonia.

In the treatment of pyæmia, or erysipelas, few persons consider the mere local affection so much as the relief of the general condition, or attempt to cure the peritonitis of pyæmia by depletion and mercurial preparations. The cases of puerperal peritonitis were nearly all of that class in which pus could be detected in the uterine veins, in the broad ligaments, or in the pelvic cellular tissue. In instances where other serous membranes are acutely affected, the symptoms are often of such a typhoid character as to preclude the use of the remedies just referred to; and those remedies best calculated to relieve the local affection are the best adapted to promote the removal or subsidence of the general disease.

The *third* and last class comprises peritonitis of a general character, arising from *struma* and *cancer*, in which the peritoneal disease is often very insidious. We include in this division those cases in which, after a prolonged state of hyperæmia, the serous membrane becomes thickened, and a very slight cause suffices to produce acute disease.

The forms of inflammatory disease of the peritoneum in *struma* vary both in degree and in extent. Sometimes tubercles exist partially distributed, as on the peritoneal aspect of ulcers of the intestine; or they are found more generally upon the serous membrane; but we have not enumerated these, unless associated with other indications considered as proofs of inflammatory change.

These tubercles were, in twenty-two cases, combined with the effusion of lymph, and the ordinary signs of acute disease. In forty-eight the changes were more chronic, and the obliteration of the serous membrane occasionally resulted from the organization of the product poured out between the coils of intestine. Acute mischief had sometimes supervened on the old disease, and lymph or pus became effused amongst the united intestines; and in several the pus had gradually found an outlet by the perforation of the intestine. In one of these the whole calibre of the bowel was divided, so that several truncated loops entered into one fæcal abscess. Firm peritoneal adhesions do not always form, and then the perforation of a strumous ulcer of the bowel leads to sudden and fatal peritonitis. Eight cases of this kind have been previously enumerated. A few tubercles were sometimes associated with considerable serous effusion, constituting a very intractable form of dropsy, especially when occurring in the young; in others, the intestines were distended with flatus to a great extent, without the pain of ordinary peritonitis.

These cases of strumous disease of the peritoneum were occasionally found with perfectly healthy mesenteric glands, although this was not generally the case: in young subjects they were often associated with tubercles in the pia mater; and in many others with phthisis. The ages were very diverse, and although many occurred early in life, an equal number was found in persons from thirty to forty years of age.

We believe the same rules ought to be borne in mind in the treatment of acute peritonitis in *struma* as in acute pleuro-pneumonia. Opium is of value, not only in relieving the pain and the great nervous prostration so constant in disease of the abdomen, but it also facilitates the recovery of the injured structure. Warmth, anodyne applications, the use of local depletion, may be added; purgatives should be avoided; and we think that mercurial preparations given so as to affect the mouth are as injurious in this form of struma as in any other; while it appears scarcely necessary to resort to this remedy for the purpose of preventing opiates from checking secretion, or to act as aperients.

In the more chronic forms of the disease, we believe the means best calculated to remove that which is local are those suited for the removal of the general state; such as nourishment as far as it can be borne, cod-liver oil, steel, as the iodide, steel wine, the iodide of potassium, alkalies, &c., occasional counter-irritants, &c.

Cancerous disease is frequently the cause of peritoneal effusions, both of lymph and of serum. To twelve of these cases we have already referred, in connection with some of the forms of perforation of the intestine; but forty others are found amongst our number, nine in males, and thirty-one in females. With very few exceptions, many other structures or glands were affected; thus, in men, the liver, stomach, pancreas, or kidney; in women, the ovaries or uterus were in the majority also implicated; and in earlier life, among the latter, and where the cancerous disease was of a medullary character, the peritonitis was generally marked by the effusion of lymph to a considerable extent. But in women, when the functional activity of the ovaries has ceased, and in men, when there is general diminution of functional power, a more chronic form of disease is set up, consisting of an immense number of small tubercles, about the size of split peas, and of different degrees of vascularity, covering the whole of the peritoneal surface, with intervening lymph in greater or less degree, and generally with dropsical effusion. This kind of disease is much more common in women than in men. Nineteen occurred in women, and only one man was affected in the forty cases we have referred to. In women the average age of the nineteen cases was fifty-two; the man was aged seventy. The other instances of cancerous tubercles upon the peritoneum in men were associated with more important glandular disease. In some, the evidence of a cancerous character in the elements of the tubercles was well marked by the structure of the cells; in others, however, there would be much difficulty in drawing a precise line of demarcation between these tubercles and simple fibrinous cellular deposit.

This kind of disease is, however, always associated with enfeebled power, and diminished functional activity. Our remedies, as diuretics, have very little effect, and any measure which still further diminishes strength appears to increase dropsical effusion. To sustain the powers of life by every available means is the best preventive against this result. If paracentesis be performed, temporary relief may be obtained; but more frequently the patient very rapidly declines, and we then find that the whole of the diseased peritoneal surface has increased in vascularity, some of the tubercles are reddened by congested capillaries, or recent lymph is poured out amongst them.

The only remaining cases are those in connection with chronic disease of the liver and thoracic viscera, producing prolonged hyperæmia of the peritoneal vessels, and ascites, and readily terminating in acute peritonitis; hence the danger of tapping in these instances; but acute peritonitis often occurs in cirrhosis from other very slight exciting causes.

In *cirrhosis* the peritoneal covering of the liver is very generally thickened. We do not, however, refer to this merely partial affection, but to those in which the serous membrane is generally affected.

Thirty-two were cases of peritonitis connected with chronic hepatic disease. Of these, in fourteen the peritonitis was chronic, in six acute and chronic, and in twelve acute disease only was found. Five of the thirty-two were rendered acute by tapping, and have been enumerated amongst operations; but in several of the remaining cases it was probable that other causes produced acute aggravation; thus three were associated with pneumonia.

Many of these instances were found in persons of intemperate habits; the arteries were often diseased, the kidneys granular or atrophied; and these facts should be borne in mind in directing the treatment. At an early stage, where the diet can be regulated, and the excretory functions of the liver, the kidneys, and the skin stimulated to increased action, the symptoms may in a great degree be alleviated; and where acute peritonitis is set up with cirrhosis at an early stage, we know of no class more benefited by the judicious use of the ordinary remedies for peritonitis, namely, local depletion, and mercurials with opium, on account of the stimulant effect mercurials have on the excretory glands; but we think that all the good effect of mercury may be attained without that remedy being used so as to produce salivation. If the peritonitis be of a chronic form, and associated with advanced cirrhosis, our measures will at best be only palliative. Some have recommended mild mercurial salivation before tapping, to prevent the supervention of acute symptoms; but we have no experience of such an application of this medicine, and believe that if tapping be really necessary, mercurial salivation would be detrimental, and increase the exhaustion which often follows the operation, or that the mercurial cachexia would lead to the speedy reaccumulation of the fluid. Mercurial frictions are less objectionable when used with moderation; and minute doses of blue pill, with tonics, as quinine, or with aperients, are, in many instances of chronic peritonitis from hepatic disease, of great service. Many other remedies are occasionally of much value in this form of disease, whether accompanied with dropsical effusion or without it, as iodide of potassium, alkalies, &c.; but to enter into all the remedial means available in the complications of cirrhosis is foreign to our purpose.

Nearly the same remarks apply to the peritonitis coming on in the course of chronic disease of the heart and of the lungs, of which nine cases were found amongst the numbers recorded.

In the second and third divisions of cases, the causes were as follows:

From Bright's disease	63
,, pyæmia, 13; erysipelas, 5; puerperal fever, 10; with pneumonia, 3	31
,, strumous disease	70
,, cancerous disease	40
,, hepatic disease	27
,, heart-disease	9
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In the foregoing analysis, we have sought to establish the following conclusions—

I. That peritonitis is never idiopathic in its origin, and that we do not find any such instance as acute disease of the peritoneum coming on from mere exposure to cold. In such supposed cases the cold merely tends to render acute an already-existing morbid state.

II. That the consideration of the origin of the disease, either in a local or general source, is the best guide to treatment, whether it arise—1st, from extension of disease from adjoining viscera, or from perforation and injuries; 2d, from blood-changes, such as occur in albuminuria, pyæmia, and erysipelas, &c.; 3d, from almost imperceptible changes or deficiencies in general nutrition modifying the state of the general health, as in struma, cancer, and climacteric changes, or where the hyperæmia of the peritoneum, consequent on cirrhosis and chronic disease of the heart and lungs, is followed, under very slight exciting causes, by acute mischief.

III. That in the first form perfect rest, the avoidance of food as far as possible, and the opiate mode of treatment recommended by Dr. Stokes in producing rest to the intestinal canal and to peristaltic action, and diminishing the collapse and prostration consequent on the disease, is the best mode of treatment, using, as far as need be, other means, as anodyne applications, warmth, local depletion, &c.

IV. That where peritonitis is a symptom of blood-change, as in albuminuria and pyæmia, it may be best relieved by the treatment of the primary disease, but that here opium is sometimes of great value, and more effective without than with mercurial combination.

V. That in the treatment of the third class the consideration of the cause is also our best guide; that strumous and cancerous disease should be regarded in their general relations; and in those connected with hepatic disease, where mercurials are sometimes of great service, the remembrance of the condition prior to the supervention of the peritonitis should prevent us from the use of means calculated to increase the primary mischief, namely, the cirrhosis, and the arterial disease so frequently associated with it; and that any benefit due to mercurial action may be attained *without salivation.*

VI. That in general the benefit ascribed to mercury in the treatment of peritonitis is not established, and may, perhaps, be correctly attributed to the opium with which it is combined.

We submit to the Society this analysis of cases, imperfect as it is, but drawn up with much care. The facts must remain, however the opinions deduced from them may be controverted.

BRIEF NOTES

OF A

VISIT TO THE LEPER HOSPITAL

AT

GRANADA.

BY

JOHN WEBSTER, M.D., F.R.S., &c.

Received Jan. 2d.—Read Jan. 10th, 1860.

Having had an opportunity of inspecting, during a recent holiday excursion in Spain, the ancient Leper Hospital at Granada, originally founded by Queen Isabella about three centuries and a half ago, after that celebrated city was wrested from Moorish dominion, I am induced to bring before the Royal Medical and Chirurgical Society some facts regarding the malady for which the institution just designated was first established; more especially, as leprosy has now disappeared from most of those countries where formerly it was so common, both during the early Middle Ages, and even at the period of the Crusades. Believing, also, that few practitioners in England of the present day, have, like myself until last autumn, ever seen unequivocal cases of a disease so frequently described in various old medical authors, and particularly often alluded to in Scripture, I trust that my present rather brief communication will not be considered by the Fellows as altogether uninteresting, although it merely contains several cursory remarks, based upon data derived from personal observation, when visiting the Granada hospital for lepers last September.

At that period, the establishment contained 53 inmates; 39 being male, and 14 female patients; showing that the former sex greatly predominated in number over the latter. This excess of male lepers has always prevailed, and ample experience proves that men are more frequently affected than women, in the proportion of about five to two at the least, speaking generally. Again, as to age, among the fifty-three leprous sufferers then congregated under one roof, there might be recognised individuals varying from a girl fourteen years old, and a lad not much more advanced, to persons of both sexes verging towards their grand climacteric. The complaint itself, under which the inmates laboured, had likewise attained a variety of stages; some patients being only recent victims, whereas others had been several years attacked; while all seemed slowly, but surely, advancing to the same result, namely, a fatal termination.

Among the patients now enumerated, a few had merely darkish-looking eruptions on their skin, of small diameter; many presented tubercular elevations on the face, forehead, about the ears, or exhibited other true marks of leprosy on their necks, arms, and hands. These parts of the body being usually the points where the malady was most frequently manifested; in fact, wherever the person's cutis became most exposed to atmospheric action. In numbers, the countenance appeared much distorted by excrescences or nodules on the nose and lips; besides which, the cheeks were, in several, so swollen as to alter entirely the features, and thereby to impart to the individual so affected even an animal-like expression. Other cases more advanced, had the mouth and tongue ulcerated, sending forth a horrible stench; their voice often becoming weak, husky, or otherwise seriously impaired, as to tone and strength, so that they were only able to speak in a whisper; while some had lost an eye, or part of their face by extensive sores, which, in particular examples, really seemed cancerous. Indeed, various instances actually noticed might be justly classed in that category. Several had lost fingers, toes, and even a hand, in consequence of ulceration of these parts; whereby the poor sufferer became a horrid spectacle of mutilation and helplessness. Two or three were all but bedridden from their physical sufferings or debility; and two formed such a mass of bodily corruption, that it would be difficult, and certainly painful, if not useless, to attempt giving any description.

It not being proposed to detail in the present communication the usual pathognomonic symptoms characterising leprosy, as observed among inmates of the Granada hospital, since the malady there seen was strictly analogous to that prevalent during former eras, I shall not occupy further time with even a short outline. Nevertheless, in reference to one peculiar phase of the mental faculties noticed in leprous patients, I may state that, *almost universally they seemed happy, and even quite contented with their really sad condition.* Some were also reported to be often singing, or attempting to do so; others laughing, and making merry, both when idle and when occupied in their work-rooms. In the central enclosure, where many male patients had congregated together, while I visited that part of the institution, several were gaily dancing to their companions in misery, while one of them played on a musical instrument; at the same time, different groups of busy talkers and hilariously disposed persons seemed enjoying themselves in various ways. The sight then witnessed was remarkable; and to observe many of the inmates thus pleased and contented with their lot, although labouring under a malady which would certainly prove fatal, after a longer or shorter period of bodily suffering, was not the least curious peculiarity noticed among the residents in this hospital.

Moreover, I may add, the Director confirmed all that is now stated respecting this usual, but singular, contentment expressed by many leprous patients; and on my questioning several respecting their condition, or the prevailing feelings they experienced, I nearly always got similar answers. One said she was very comfortable; another replied he had everything that was required; and a third, who accompanied on the guitar a ditty he then tried to sing, answered my inquiries by saying, "Why should he not be pleased? having nothing to complain of, but much to make him thankful." Such a peculiar feature in this disease like that now mentioned, from being often present, is very interesting, and therefore deserves record; while having been likewise generally observed among inmates by the superintendent of the Granada establishment, no doubt can remain regarding its frequent existence.

Another important observation, made by the same gentleman to myself, during our lengthened conversation, equally deserves being specially stated, namely—that he had found many leprous patients very lascivious in conversation, and that they would often become so in their conduct, were great care not always taken to keep the sexes separate. This he had observed after much experience. Indeed, that authority further added, the "*libido inexplebilis*" noticed by various ancient writers, still constitutes a prominent symptom among numerous victims under his superintendence.

With reference to the particular districts from whence most inmates received at the Granada hospital for lepers usually come, it may be stated that very rarely any are natives either of the city itself, or belong to the adjacent "Vega," which has for many ages continued so celebrated on account of its rich soil and abundant productions; being, in fact, one of the most fertile regions throughout Spain. Almost all the patients admitted are inhabitants of places adjoining the low, south-eastern Mediterranean seashore, especially in Almeria, Adra, Motril, Malaga, Velez-Malaga, or other towns on that coast, and adjacent villages —Cadiz and its vicinity being also affected. The malady would hence seem as if especially obnoxious to marine populations, not those persons who dwell habitually upon more elevated or inland localities. Recent observers likewise further assert, confidently, that leprosy is now an increasing malady, as to number, in various situations of the Peninsula; more victims of the disease being at present recognised, than during the early part of the current century. Such an assertion, however, requires additional proof ere being admitted implicitly.

Nevertheless, that the complaint still prevails to a considerable extent in some regions of Spain, may be conclusively illustrated by an instructive fact recently communicated to the Madrid Royal Academy of Medicine by Senor Mendez Alveiro, viz., that 284 leprous patients were ascertained to be alive during 1851 in nine Spanish provinces, without reckoning many more about whom no statistical return had been procured, at that date, from the districts where they resided. The above authentic statement of Dr. Alveiro fully demonstrates how frequently this loathsome disease still continues to prevail throughout the country.

Respecting the 284 leprous patients so reported, it seems interesting to mention that 188 were males, and only 96 females; giving two of the former to one of the latter sex. Further, it also appears worth stating, that 221 of the total number ranged from fifteen to forty-five years of age; consequently, about three fourths of the whole were persons of middle age, or during full manhood. Among the males, 96 were married, 86 bachelors, 4 widowers, and one an ecclesiastic; while of the females, 52 were single, 32 married, and 12 had become widows; thus showing that 139, or almost one half, were unmarried when affected with leprosy. These figures, therefore, conclusively bear out the statements enunciated in a previous paragraph of this paper, which alluded to the sex and age of the customary sufferers from the malady under discussion.

On the other hand, if the frequency of leprosy in Spain at present be compared with its actual occurrence in the United Kingdom, the contrast becomes both curious and instructive; the malady of former epochs has now, it may be fairly asserted, all but disappeared from the British Islands, among persons who have never visited any tropical climate where it rages endemically. One of the latest illustrations of this complaint affecting a native of the British Empire, and coming within that category, occurred in the Edinburgh Infirmary during 1809; while two cases were also, it is said, recognised several years ago in one of the western islands of Scotland.

Through the kindness of Dr. Addison, I have, however, been informed that he had met with an unequivocal example of true tubercular leprosy in a man under his own care at Guy's Hospital, who was never out of Great Britain, and therefore constituted a case really indigenous. Dr. Wilks has published, in 'Guy's Hospital Reports,' an account of this remarkable instance of "lepra tuberculosa," drawn up by Dr. Gull, from which he courteously permits me now to quote the following idiopathic and salient features. This patient, aged 28, worked as a tailor, and was born at Cork, which he had left twelve years before his admission into Guy's Hospital; five of these years having been passed at Croydon, and the remaining seven usually in London. Until eight years prior to his being treated by Dr. Addison, at which period the first symptoms of true leprosy appeared, this patient had always enjoyed good health, with the exception of having twice contracted syphilis, although in a mild form. All treatment proved unavailing, while the disease continued slowly to make manifest progress, and to exhibit every characteristic of this hideous malady, until the 24th of May, 1853, when he died, after having actually walked out of doors as usual on the same morning.

In reference to the above case, whereof drawings were exhibited before the meeting, through the kindness of Dr. Wilks, and which is the most recent example of unequivocal leprosy I have heard recorded as actually affecting any native of Great Britain who never visited or resided in localities where that disorder prevailed; it seems further worthy of mention, that he neither experienced any change in his sexual feelings, nor manifested the singular hilarity so frequently noticed among many leprous inmates under surveillance at the Granada hospital.

Indubitably, examples of this malady which exhibited an unmistakeable character, have fallen under the observation of other medical practitioners in England, among persons coming from, and natives of the West Indies, or countries devastated by leprosy; while it sometimes also attacks Europeans who have resided during successive years in situations where the disease is endemic. However, according to my present knowledge, no one has recently met with a case in Great Britain really indigenous. That fact becomes much more worthy of remark, when it is remembered how very common this malady was reported to be in the British Islands during former ages, even among the highest classes of society; as, for example, King Robert the Bruce, who, history reports, died its victim after several years' sufferings. Besides, it should likewise be recollected, that not many centuries ago, there existed upwards of one hundred lazarettos in Scotland and England; one of these, at Sherborne, near Durham, containing usually sixty-five inmates. Nay, it is also stated, that St. James's Palace in London was originally built as a receptacle for lepers.

Hereditary tendency is another point to which I would now beg to draw attention; since it is believed by some medical observers to exert considerable influence in producing leprosy. Supported by the statements made to me at Granada, this opinion seems very doubtful, if not erroneous. Indeed, among a large number of the patients there resident, no proof of any transmitted affection could be actually traced. On the contrary, their relatives were often reported to be both healthy and free from leprous indications. It should, however, be remarked that many of these inmates had lived, and were actually born, in districts where leprosy prevails endemically, and in which they were exposed to the influence of similar insalubrious causes, whether of soil or atmosphere. Further, numbers had also suffered from the effects of defective diet, and lived in unhealthy habitations, besides being frequently victims of filth and abject poverty. In fact, this most loathsome of human physical infirmities, considered with reference to its production, according to my own observation, seems analogous to cretinism, goitre, pellagra, or ague, and may be fairly classed in the category of endemic diseases.

As an illustration that hereditary predisposition does not often or usually prevail, I would here allude, among other examples, to the cases of three brothers seen in the Granada Leper Hospital, who were each simultaneously affected with leprosy, and respecting whose history I then made special inquiry. In these respective instances, although the malady was unequivocally marked, but still exhibiting different stages, no sister, other brother, neither parent, nor any relative, had ever manifested indications of leprosy. Nay, more, they all were reported healthy individuals, and perfectly free from that complaint. On this question a curious example may be quoted, which occurred in another district of Spain, viz., that of a healthy father married to an undoubtedly leprous female, by whom he had no children. Having espoused, after her death, another wife, who was free from any taint, but lived in the same locality, he had by her four sons: of these three ultimately became lepers, notwithstanding the disease had never manifested itself in either parent. Therefore, other causes must have come into operation, and produced the complaint. It may, however, be here specially mentioned, as perhaps the fact will partly explain the supervention of leprosy in certain families, that this scourge, which is frequently now called elephantiasis on the opposite African coasts, not only often there prevails among natives, but frequently attacks, it is said, the descendants of persons originally from other countries, and whose progenitors had resided in these regions consecutively during, at least, three generations.

Another feature should likewise be specified, which usually characterises the true leprosy observed in Spain—at least, such was the information I received when making inquiry—namely, that the malady seldom, if ever, affects persons belonging to the middle or upper classes of society, but almost invariably attacks the poorly fed and badly lodged labouring population. This result has been particularly noticed in dwellings that are damp, devoid of free ventilation, and whose occupants sleep on the ground floors, or in the same hovels with mules and donkeys, frequently wallowing among ordures, besides suffering from all the evils of indigence. Therefore, its absence in recent times from localities which were formally afflicted may be held as decisive indications of advanced civilisation in any country where this loathsome, incurable disease proved at one time so rife and destructive among residents.

Respecting those causes which appeared often to exert the most influence in producing leprosy throughout populations situated under the local circumstances already briefly adverted to, it would appear not only that innutritious diet acts powerfully, but even the kind of food which is consumed under such circumstances exerts a baneful influence. Thus, putrid fish, whereof residents near the sea-coast oftener eat than persons dwelling inland, may, to a certain extent, explain the greater frequency of this malady in maritime districts, compared with central and more elevated regions. Besides such deficiency of proper nutriment, eating diseased hog's flesh, habitual intoxication produced by deleterious "aguardiente" (bad, fiery brandy), consumption of old, mouldy grain, a deficient supply of vegetables, the want of salt, and the scarcity of, or often total abstinence from, animal food as an article of diet among the poor, prove equally most injurious. Whereas, throughout such fertile inland plains as the productive Vega of Granada, where the labourers are better fed, besides otherwise enjoying greater physical advantages, both of situation and climate, than the seaward residents, leprosy is much less observed.

Before taking leave of the etiology of the specific disease affecting residents in Queen Isabella's hospital at Granada, I would add, in reference to these moot questions, according to the experience of a medical friend and Spanish physician, who courteously accompanied me when visiting this establishment, and from whom I obtained much information, true leprosy cannot be correctly designated infectious, although that gentleman admitted it was contagious. Or, like syphilis, variola, and the vaccine disease, the malady may be communicated by inoculating another person with matter taken from an individual already affected. So far this authority considered leprosy might be propagated from party to party. Both here and elsewhere, it is often confidently stated by writers, that the ordinary attendants at leper-houses rarely, if ever, manifest any symptoms of the disease affecting inmates, notwithstanding they may have long resided in such establishments. Besides which, competent witnesses also acknowledge that leprous victims may remain at home for years, without infecting any other member of their family, or individual with whom they reside. From such evidence it would therefore indubitably appear proved that leprosy is not an infectious disease, according to the ordinary meaning of the term, but may be, nevertheless, communicated by virus taken from persons attacked, and hence, strictly speaking, like smallpox, is really contagious.

Previous to closing these cursory statements regarding a malady now very seldom met with in any part of Europe, except Norway and Greece, where leprosy still devastates the maritime population of these districts, and occasionally also on the southern coast of France, near Marseilles, besides various places in the Spanish territory—about which, the present observations are mainly founded on data obtained during my late visit to Andalucia—it only remains to make a few succinct but general remarks respecting the medical treatment, as also the hygienic mode of managing leprous patients I recently saw in the hospital at Granada. Upon this important question, although no expectation can be reasonably entertained of curing confirmed cases by remedies, or even of arresting their fatal progress, after a certain stage, still something may perhaps be occasionally accomplished towards alleviating the sufferings of those victims who have become martyrs to such an inveterate and incurable disease as the one in question.

At the institution here passed briefly under review, through attention to diet, cleanliness, frequent bathing, and proper clothing; and the judicious employment of patients in manual occupations, according to their physical strength and individual capacity; by their remaining much in the shady court-yards or adjacent garden, where refreshing, pure air may be breathed, aided by varied recreations, good effects towards ameliorating the sad condition of inmates may be sometimes, at least, obtained. In short, the system pursued mainly resembled that now followed at most well-regulated lunatic asylums. Of course, various local remedies were applied to ulcerated surfaces, and usually soothing applications; while the physician also prescribed medicines internally, according to symptoms, chiefly of a tonic description. But in regard to anticipating any permanent beneficial result through medical treatment, such an expectation was almost invariably considered nugatory; I therefore need not here enter upon so useless a discussion. It should, however, be added, with respect to the effect of remedial measures, that after leprosy has become decidedly pronounced in any patient's constitution, the disease rarely continues longer than eight or ten years before it terminates fatally. In support of this assertion, I may again refer to the 284 leprous persons which were previously quoted; of these, 79 had been affected, at the time they were officially enumerated, from one to five years, and 122 from five to ten years. The remainder were of much longer duration; one having been thirty years a sufferer, while another is said to have actually laboured under this disgusting complaint during thirty-five years consecutively.

The hygienic management of inmates usually pursued at the Granada institution next requires some passing remarks. There many patients appeared zealously employed in making various kinds of matting with "esparto," a species of rush, which serves for numerous domestic purposes throughout Spain. Among other uses to which it is applied, that of sacking to carry farm produce may be named; but especially in manufacturing baskets and panniers for mules and donkeys. Hence, these varied kinds of occupations become an extensive source of profitable manual labour to the labouring population of the Iberian Peninsula. Some leper residents were likewise occupied at different handicrafts, such as shoemaking, tailoring, and so forth; while in the female division, the laundry or kitchen required the services of several; besides others who acted as domestic servants in different departments of the institution. Attendance at chapel, and the brief customary religious ceremonies regularly performed therein, also constituted a frequently recurring source of employment and satisfaction to inmates, whose monotonous existence was thereby agreeably varied, and their feelings, as Catholic Spaniards, essentially gratified; to say nothing of various other important beneficial considerations.

While alluding to the prominent part which duties of the sacred description now specified always play in every benevolent receptacle of Spain, having been present at the Leper Hospital of Granada when nearly fifty patients were assembled in its chapel at the same time, with their faces all turned in one direction, during the devotional services then proceeding, whereby they could only be observed by spectators, never by each other—and further, the sexes being congregated in separate apartments—it may be here remarked that, at no public establishment of any kind ever previously inspected throughout Europe, have I witnessed such a disgusting sight as the spectacle these miserable and afflicted fellow-mortals there presented to the eyes of professional or curious visitors. The disfigured, ghastly countenances of some, covered with deep, extensive sores; the distorted features of others; the idiotic expression of many; and the feeble physical frames of most; at the same time that every person was then devoutly kneeling and engaged in prayer; altogether formed such a melancholy exhibition of frail humanity, that the scene baffles description, and I have no desire ever again to join in any similar ceremony, or one so lugubrious.

In concluding the present short memoir respecting the Leper Hospital at Granada, readers will, I hope, permit me to observe, that from the several statements and facts contained in previous paragraphs, all obtained from reliable authorities,—the following general inferences, if not fully established "dicta," may be legitimately deduced.

1st. Leprosy chiefly affects the male sex, as it always has done heretofore.

2d. Every age is liable to its attacks, but mostly that after puberty and during manhood.

3d. The malady is not infectious, in the strict sense of that definition.

4th. It seems to be an endemic disease.

5th. Occurs only among the lower and badly fed ranks of society, at present.

6th. Residents on the sea-coast constitute its ordinary victims in Spain, as elsewhere.

7th. The principal apparent causes seem to be putrid, indigestible, and innutritious aliment; also indigence, filth, and occupying insalubrious dwellings in marshy, miasmatous soils; or where a humid, variable, marine atmosphere prevails.

8th. Leprosy may be communicated by inoculation,—according to some authorities.

9th. It is incurable when fully developed; hence in the latter stages, all medical treatment proves unavailing.

10th, and lastly. Although the disease seems nearly extinct in districts where leprosy formerly prevailed extensively, and has almost disappeared from every other European country excepting Greece and Norway, it yet still exists to some extent throughout Spain; especially among poverty-stricken natives, who live either upon or near the southern Mediterranean sea-shores of that Peninsula.

OBSERVATIONS ON STERTOR,

AND ON THE

VARYING CONDITIONS UPON WHICH IT IS DEPENDENT,

WITH THE TREATMENT NECESSARY TO ITS RELIEF.

BY

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COMMUNICATED BY

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In order to make my subject clear, I am necessarily led to speak of obstructions about the throat in connection with stertor; how far these obstructions are due to the position of the body, and how they may be removed by altering that position.

The cause of the loud and deep sound in the throat to which the name "stertor" has been applied, does not appear to have received the attention it deserves. From numerous experiments I have arrived at the conclusion that in the majority of instances it arises from one of three conditions: from paralysis of the velum palati; from the paralysed tongue falling back in the throat; and from the presence of mucus in the pharynx and air-passages.

In the winter of 1855 and 1856 I was engaged, in conjunction with Dr. Edward Long Fox, of Bristol, and Mr. Charles Hunter, of Wilton Place, upon some experiments for the late Dr. Marshall Hall, to determine the amount of air which could be respired in the dead subject by compression and relaxation of the chest. In attempting this we repeatedly found that at one time compression failed to expel any air from the chest, and at another, after air had been expelled, it would not re-enter on removing the pressure. On opening the mouth and searching for a cause, it was found that the tongue had fallen back and blocked up the cavity of the pharynx. The tongue was accordingly drawn forward and fixed between the incisor teeth, but still we found that on one body the experiment succeeded well in producing respiration, in another it failed. This time the tongue was no obstacle, but the pressure on the chest and abdomen had thrown up some of the contents of the stomach into the pharynx, which entirely prevented the current of air from passing into or out of the chest. This induced us to try to produce respiration in such positions that anything in the pharynx would fall *from* rather than *towards* its posterior wall; the consequence was, that we obtained the most invariable and favorable results by the method of artificial respiration now known as that of Marshall Hall.

In a paper communicated to the Medical Society of St. George's Hospital in 1856, great stress was laid on the influence of the position of the body over the tongue in the half-drowned patient; and it was pointed out how necessary it became to pay the greatest attention to a fact which at first sight appeared of little importance. Subsequent to the meeting I received from Mr. Harvey, one of the members of the Society, a letter on the subject, of which the following is an extract:

"I am glad to find you lay so much stress on the tongue's position producing apnœa in persons that have been rendered insensible. One of the first persons I ever saw on the operating-table nearly died from this cause during the operation, under the influence of chloroform. Mr. Syme, who was operating, fortunately guessed what was the cause of the sudden asphyxia, and drew the tongue forward with a pair of forceps, and the patient presently recovered."

Mr. Harvey's case strikingly illustrates that the tongue may be a cause of apnœa in other circumstances than those of drowning. The following cases illustrate another phase of this subject :

Case 1.—In November, 1857, I was called to see an old lady who had been attacked with sudden sickness whilst talking to some friends; faintness followed, with an inability to express herself, though she was to a considerable extent conscious. She was put to bed, and kept quiet; mustard poultices were applied to the epigastrium and nape of neck, and hot bottles to the feet. In an hour or two reaction commenced, and at the same time coma and stertorous breathing set in, which steadily increased in intensity until my second visit, at which time this condition had lasted about three hours. Her pulse was now full and excited, but as she was seventy-three years of age, and had already suffered from two or three attacks of paralysis, no severe measures were adopted. The skin was hot, and covered with a profuse perspiration; the face was swollen and livid, and the mouth half open; the pupils were contracted and insensible to light, and the eyeball to touch. The stertor was very deep, and the breathing laboured. Whilst I was at the bedside of my patient she was very nearly suffocated, from the stomach ejecting a portion of its contents into the throat. I at once turned her on her side and emptied her mouth, when she not only breathed freely, but without the slightest stertor. I was somewhat surprised, but proceeded to place her in what appeared to be the most comfortable position, *on her back*. Immediately when I did so, her respiration suddenly ceased, and she convulsively struggled for breath. Supposing it to be the tongue which had choked her, I thrust my finger into the mouth, and pressed it forward, when the breathing again became easy and free from stertor. The patient was now comfortably laid on her side; and until her death, which took place two days afterwards, the stertor never recurred when she was in that position, but the respiration continued as naturally as if she slept. To convince myself that this was due to position, I several times turned her on her back for a few moments, and invariably, when I did so, the stertor was as marked as ever. The lateral position had another advantage, for it allowed the saliva and mucus to drain away instead of remaining in the bronchial tubes and giving rise to "mucous stertor," and thus offering another impediment to the respiration. Other changes occurred coincidently with the change of position; but how far they depended upon it, future experience must decide. The pulse became calmer; the skin cooler and less bedewed with moisture; the eye sensible to the touch; and general sensation returned to such an extent that the patient evinced signs of discomfort at the removal of a mustard poultice. Nevertheless, whenever the supine position was adopted, the stertor, difficulty of breathing, lividity of face, and other unfavorable symptoms, returned in full force. It was easy to observe, that as the position was changed, the paralysed tongue gravitated downwards.

This case suggests many important points for consideration:—1st. That the paralysed tongue *may*, under certain circumstances, cause even death by suffocation; for I believe this patient would have died if I had not been present when the tongue fell so suddenly back, and when the fluid was ejected from the stomach into the throat. 2dly. That stertor arises from the tongue falling back in the supine position of the body, thus offering a serious impediment to the respiration; since it required greater muscular force to breathe with stertor than when the respiration was noiseless. 3dly. The mucus (another respiratory impediment) drained away when the patient was on her side. 4thly. The great improvement of the general symptoms occurring upon the establishment of easy breathing in the lateral position: *pulse, skin,* and *sensation* all undergoing material alterations for the better.

Case 2.—G. T—, a gentleman 65 years of age, became comatose from exhaustion supervening upon extensive subacute inflammation of emphysematous lungs. When I entered the room, he was lying on his back, with his mouth open, breathing stertorously, though not to a great extent. On looking into the mouth, the soft palate appeared remarkably small and shrivelled, so that it did not come very decidedly in contact with the tongue, even though partially paralysed. Whilst I was standing at the bedside, my patient suddenly ceased to breathe, became very livid in the face, struggled convulsively, and then became rigid. My recollection of the first case induced me to turn this patient on his side, when he instantly recovered with a long groan, and then breathed quite naturally. He was turned on his back again; stertor returned, and he became choked from a quantity of bronchial secretion. The lateral position was resumed, the sputa drained away, and he now breathed quite easily until his death, which took place about half an hour afterwards.

Case 3.—H. J. L—, a clergyman, about 60 years of age, subject to slight epileptic seizures twice a year, was seized, on May 4th, 1858, at ten in the morning, with a fit of the usual character. The attack commenced with a scream, and lasted but a few minutes, though the patient was some time before he fully recovered. At eight o'clock in the evening of the same day, he was again attacked whilst walking across the hall of the hotel at which he was staying. When I arrived about ten minutes afterwards, I found him lying on his back, with a deep cut over the left eye, breathing stertorously, and with much labour. The mouth was open, the face highly congested, the skin hot and perspiring, and the pulse full and sharp, but not accelerated. He was quite insensible to external impressions. He was turned on his side sufficiently for the tongue to fall forward; the stertor instantly gave place to easy, natural breathing; the congestion of the countenance gradually disappeared, and in *less than one minute* he began to evince signs of recovery, and almost as quickly the pulse became weak and small, and the skin cool. He went to sleep for a few minutes, and on awaking was rendered almost frantic by pain in his head. From this he slowly recovered, and in a few days was convalescent. I should mention that there was a loud bruit at the apex of the heart, and that a small pulse was common to him.

Case 2 merely gave further evidence of the danger of the supine position of the body from paralysis of the tongue and the accumulating bronchial secretion; but in Case 3 it would appear that the change of position was in some measure the cause of his speedy recovery. In this case, too, as in Case 1, the skin and pulse altered in character as the stertor ceased. It is in such a case as this that I can imagine "position" to be a curative as well as a palliative measure. Here was a delicate man, subject to epilepsy and disease of the mitral valves. Supposing the breathing to have gone on with great labour, and the lungs to have become more and more congested, and consequently the brain and heart also, one or other, or both of these vital organs, might have suffered, and death ensued.

Case 4.—Mrs. P—, æt. 46, after suffering twelve days from continued fever, was seized on the evening of August 27th, 1858, with a severe rigor, which was soon followed by what was supposed to have been a very deep sleep. I was called to see her at two in the morning of the 28th, when I found her lying upon her *side*, in a state of deep coma, with her mouth half open. There was no stertor; the skin was warm and bedewed with moisture, and the pulse was almost imperceptible. I endeavoured to administer a little stimulant, and for that purpose turned the face upwards, when I soon perceived a harshness in the breathing, from obstruction in the back of the throat. By allowing the head to remain in this position a few minutes, the obstruction became greater at each inspiration, and *stertor* was the result. A little fluid was now introduced into the mouth with a teaspoon, which almost choked my patient; but on turning her on her side again, the fluid drained away, and the stertor ceased. In about an hour afterwards she sank.

So prostrate was this poor woman from fever, that I believe, had much effort been required to overcome the impediment to the respiration, from the time coma first set in, she would not have survived as long as she did.

Besides the cases now related, a medical friend informs me that, on my suggestion, he adopted the lateral position in a case of stertor from apoplexy, and that the stertor immediately ceased. The man was bled, and soon recovered from the comatose condition. My friend also adopted this method with a child suffering from convulsions. When he first saw the child, it was frothing at the mouth, and the respiration seemed much impeded. He turned it on its side, and before he had time to apply other remedies, the child began to evince signs of recovery. I do not assume in these cases, or in Case 3, that change of position was the *cause* of recovery: it may have assisted to produce the happy result, or it may merely have been a coincidence. I simply state the facts as they occurred, and avoid inductions for the present.

Meanwhile, I propose roughly to consider the anatomy of the parts concerned in stertor, and to show *how* and *why* stertor is the consequence of the supine position of the body, and why it differs in character in different cases. I must also relate a few experiments undertaken with the object of further illustrating the views herein entertained.

It is well known that the cavity of the pharynx, having a fixed boundary only posteriorly, may have its capacity materially affected by the ever-varying position of its sides, of the soft palate, the tongue, and the larynx. The larynx and sides of the pharynx have, I believe, little connection with the cause of stertor. I shall therefore not further refer to them. The tongue being attached to the lower jaw by its mucous membrane and its muscles, has its relations with surrounding parts altered as the mouth is opened or closed.

When the mouth is closed, the ramus of the jaw forms nearly a right angle with the spinal column, from which the symphysis is then at its greatest distance. When the mouth is opened by the dropping of the jaw, the symphysis describes the arc of a circle, and approaches more nearly to the spine, where the posterior wall of the pharynx is attached. (See Drawing at the end of the paper, fig. 1.) The tongue having its chief attachment to the symphysis, would consequently be drawn away from the pharynx in the former case, and allowed to rest in contact with it in the latter. This, if not true in every case, is at least true in some, as the plate demonstrates. (See fig. 2.)

The dissection from which the drawing was taken was made with great care, so as not to displace the parts more than was necessary to give a fair view of the base of the tongue and the epiglottis. The two sketches, with the open and closed mouth, were kindly taken for me on the spot by my friend Mr. Fitzgerald, and they very accurately represent the parts as they appeared at the time. When the mouth was open and the subject on its back, the road to the larynx was completely obstructed, in consequence of the tongue resting in contact with the back of the pharynx; but the obstruction could be removed by hooking the tongue forward. When the mouth was closed, the tongue was lifted from the pharynx, leaving ample breathing space. But even with the closed mouth, if the chin were much bent on the sternum, the base of the tongue was almost in contact with the back of the pharynx.

It would seem from this dissection that the muscles of the tongue attaching it to the symphysis of the jaw, are too short to admit of the base of the tongue reaching the back of the throat when the mouth is closed. The purpose served by such an arrangement would appear to be to keep the base of the tongue away from the back of the throat during deglutition. In the act of deglutition, the mouth is firmly closed, and the genio-hyoid and genio-hyo-glossi muscles taking their fixed point from the now immoveable symphysis, draw the body of the tongue upwards and forwards, allowing sufficient room for the morsel of food to travel easily through the cavity of the pharynx. Were these muscles of greater length, the base of the tongue might become a source of inconvenience, if not of danger. The dissection, besides demonstrating the respective positions of the tongue with the open and closed mouth, suggests the necessity of caution being used in raising the head with pillows; for if the head be too much bent forward on the chest, the tongue may lie in dangerous proximity to the pharynx, even if the mouth be closed.

The Different Characters of Stertor.

From having observed the snoring of sleep sometimes take place through the nostrils with the closed mouth, and sometimes through the open mouth, and having also observed differences in the apoplectic stertor, I endeavoured to analyse the particular causes of each of these varieties by some experiments on myself, and by clinical observation.

I placed myself as nearly as possible in the condition of an apoplectic patient, by paralysing my tongue, or rather by allowing it to assume its own position by gravitation. After a little practice, and with some care, I succeeded in effecting this, and found—1st. That it required much more voluntary effort to *snore* through the nose and breathe stertorously through the mouth in the upright and prone positions than in the recumbent and supine, and that the greatest impediment to the respiration occurs when the jaw is dropped and the mouth most widely open. 2dly. The deep, vibrating snore of sleep, with the closed mouth, depends upon the tongue pressing the velum palati upwards and backwards, thus narrowing the opening between the nose and pharynx, the velum vibrating as the air rushes through. Sometimes, when the mouth is partially open, the soft palate drops upon the tongue, and vibrates as the air rushes between it and the tongue. To either of these noises in which the soft palate is concerned I propose, for the sake of distinction, to apply the name of "palatine stertor." 3dly. The harsh, sharp, husky noise, occurring in apoplexy, with the mouth wide open, always arises low down in the pharynx, and depends upon the narrowing of the passage between its posterior wall and the base of the tongue. By holding a looking-glass before the face whilst producing this noise, it can readily be seen that the soft palate is in no way concerned in it. In all the cases recorded in this paper this was the nature of the stertor which existed, and as it arises solely in the pharynx, I have given it the name of "pharyngeal stertor." 4thly. There is a variety of stertor in apoplexy which may be named "mucous stertor," as it depends upon the presence of mucus in the bronchial tubes. So common is this, either alone or in combination with one or other of the preceding varieties, that in 'Hooper's Medical Lexicon' it is stated, that "apoplectic stertor appears to depend upon the presence of mucus in the air-tubes," as though it were always the cause.

Of these three different varieties of stertor, that which is named pharyngeal is far the most dangerous, for the greater the paralysis the more effectually does the tongue block up the passage, and each inspiration only adds to the distress by sucking the tongue lower down into the pharynx. Its danger is, however, not due alone to the gradual impediment it offers to the respiration, but also to the fact that at any moment this impediment may be absolute, and cause death by suffocation. This has been before referred to in the remarks upon Case 1.

We may observe a similar example in the snoring sleeper, who, after drawing several inspirations with evident effort, at last comes to a sudden check from having been gagged by the tongue falling back. This will rouse him from his stupor, and he will then sleep on quietly until the same thing recurs from his deepening sleep.

This subject gave rise in my mind to the question—Is the breathing in deep coma invariably carried on through the mouth? I once thought that such was the case. In the four cases related it was so, as well as in some cases of apoplexy which I witnessed at a former period. But the following case is one in which the breathing was carried on through the nostrils.

Case 5.—W. B—, æt. 41, had suffered for two years from numbness and tingling of the extremities of the right side, and was now (August 20th, 1858) under treatment for a supposed commencing disease of the retina. He had been suffering much from anxiety for some time past, and was unusually drowsy in the daytime. On August 20th, after eating a hearty breakfast, he was seized with paralysis of the right side. When I arrived he was able to speak indistinctly, though not to make use of the words he wished. The face was drawn to the left side, and the tongue pushed to the right when protruded; the pupils were natural. In a few minutes he ejected a large quantity of food from his stomach, and became covered with perspiration. He was conveyed to bed, but before he could be undressed he was partially sick several times. He soon commenced *snoring* with his mouth *closed,* and was now unable to answer questions; his pulse was soft and feeble, and his skin clammy. I turned him on his side, and though the snoring altered in character, it did not cease, but the impediment to the breathing was less. The mouth was firmly closed, but by means of a spoon I was able to open it slightly, and found the tongue in contact with the incisor teeth, and closely applied to the palate. By introducing the spoon further into the mouth, and pressing on the tongue, all snoring at once ceased; when, however, the mouth was allowed to close, it continued as before. Medicines could not be swallowed, and in about half an hour he breathed his last, quietly and without a struggle.

Remarks.—Snoring in this case continued even in the lateral position. The spasmodic closure of the jaw had pressed the tongue upwards and backwards, carrying with it the soft palate. The snoring was, however, soft and slight, and was but a very partial impediment to the respiration.

But I am inclined to believe that the peculiar apoplectic condition giving rise to spasmodic closure of the jaw and "palatine stertor" is exceptional; and that the proposition that "it is the *rule* for persons in coma to breathe through the mouth" is a true one, and for this reason: the muscles of the jaw being paralysed, the jaw drops and opens the mouth; whereas the dilators of the nostrils being also paralysed, the alæ nasi are forcibly drawn by the in-going air towards the column of the nose, and thus close the orifice of the nares altogether. This can be illustrated by the act of "sniffing;" but the following is a case in point:

Whilst watching the dying moments of a poor man who was comatose from hæmorrhage from an ulcer of the stomach, I observed that he breathed with evident and increasing effort, from the alæ nasi flapping towards the centre column during inspiration. With my fingers I kept the nares freely opened, and the breathing became so easy that I have reason to think that his life was prolonged for some little time by these means.

General Reflections.

Having now given in detail what evidence I possess in favour of the foregoing views on "stertor" and the "influence of position in certain morbid conditions of the system," I have to add a few remarks on the cases to which this subject appears to apply, and more particularly in reference to apoplexy and its treatment.

Since the days of Abercrombie so much light has been thrown on the different causes of apoplexy, that his division of the primarily apoplectic (where no apparent change had taken place in the brain) may be broken up into several subdivisions. It is now fully acknowledged that many of these cases depend primarily on some disease of the heart or its valves, upon aneurism, upon atheroma, upon engorgement of the pulmonic circulation, and upon uræmia. Consequent upon these great advances in pathology is a total change of treatment, for it is but seldom that we now hear of that indiscriminate and enormous bloodletting which is detailed in Dr. Abercrombie's work on diseases of the brain. In his time, age, sex, or condition had little influence over treatment; so long as apoplexy existed, bloodletting was considered the chief, almost the only, remedy.

Since, then, it is admitted that apoplexy may result (and very commonly does result) from an alteration in the state of the general circulation, I conceive that anything which would mischievously affect the circulation during the apoplectic attack would militate very considerably against the chance of the patient's final recovery. Let me suppose a case. A man with disease of the heart or large vessels, on making some unusual effort, falls down in a fit; he is taken up, and laid on his back in bed. Stertor at once commences; if it be not relieved, the respiration becomes impeded, congestion of the lungs takes place, then of the right side of the heart, then of the jugulars and of the brain. And now another direct source of apoplexy exists—*increased congestion of the brain*, and this, too, with unaërated blood, itself a poison sufficient to destroy life. As long, then, as the stertor is allowed to continue, the mischief increases, and remedies will be of little avail. But suppose that when the man is taken up in a fit of apoplexy he is laid in such a position that his respiration cannot be impeded, that the blood may flow steadily on in its course; that he is placed in such a position, in short, that nature will meet with no impediment in her attempt to right the temporary disturbance which a sudden effort brought about. Then, I think, we may fairly hope for a better issue, if judicious aid be afforded by means which the urgency of the case may render necessary. This especially applies to cases of congestion dependent upon, or complicated with, atheroma; the danger would then be doubly great, for the increased congestion might be more than the diseased cerebral vessels could bear; they might give way, and the case would become one of sanguineous apoplexy.

In the second class of cases of Dr. Abercrombie (those not primarily apoplectic), where the symptoms depend upon an effusion of blood in the brain, if there be any hope, it must at once be forfeited if the respiration be impeded, since this would increase the congestion of the cerebral vessels and cause the blood to make its way out of those which were ruptured with greater force. The same would apply to laceration of any of the sinuses or vessels of the brain from fracture of the skull. I remember to have seen a boy who had fallen from a bridge brought home in a state of syncope. Extensive simple fracture of the vault of the skull was detected. He was laid on his back on a table, and as reaction commenced he fell into a state of coma, with stertorous breathing. In a short time the symptoms became so urgent that trephining was performed, whereupon a large quantity of blood made its escape with considerable force. The boy was relieved, but died three weeks afterwards.

On examination after death, there was found a large quantity of clotted and fluid blood on the surface of the brain, the superior longitudinal sinus was lacerated, and the substance of the brain much injured.

In a case of this kind, to impede the onward current of blood through the jugulars must be the first step towards death, for the blood would make its way where there was least resistance, through the wound of the sinus. If, however, there were no obstruction in its natural course, we may fairly presume that it would follow that rather than turn aside through an opening in which, perhaps, a coagulum had already formed.

Such, then, are the dangers which may result from a persistence of stertor in apoplectic and similar conditions; but there remain the other and more immediate dangers of the supine position. 1st. Fluids or other foreign matters may make their way into the larynx from the mouth or the stomach. This I had repeated opportunities of observing in the dead subject when engaged on experiments for the late Dr. Marshall Hall; but that it is a real danger in the living man is placed beyond doubt by a remarkable case which is related in the 'Medical Times' of April 23d as having been under the care of Dr. Bristowe. A drunken man died from symptoms of suffocation. At the autopsy some of the contents of the stomach were found in the bronchial tubes. Further, my friend, Mr. Bateman, of Folkstone, informs me that during his attendance on a case of delirium tremens, the patient, in an attack of vomiting, became suddenly asphyxiated, and died. No post-mortem examination was made, but it seemed probable that some foreign matter had entered the larynx, and caused spasm of the glottis and death. 2dly. The falling back of the tongue, causing sudden and complete apnœa. This may occur in all cases of paralysis, whether from syncope, apoplexy, concussion of the brain, chloroform poisoning, suffocation from carbonic acid, or drowning. I have seen imminent danger from this cause, but am unable to record a death arising from it, though this may be a more common occurrence than we are at present aware of; for if a disaster arise from any of the above causes, the patient is, I believe, invariably laid flat on the back.

As an appendix to this subject, and in conclusion, it will be interesting to relate the following case, which has recently been forwarded me by Mr. Lewis (Medical Superintendent of the Cheshire and Chester Lunatic Asylum), to whom I wrote many months ago on this subject:

"Seth Yarwood, a man with a very peculiar formation of head, the subject of ambitious monomania of two and a half years' standing. He first had an epileptic fit, which was of a very severe character, on the 21st of November, 1858, and another on the 10th of December following. They did not recur until June 3d of the present year, when he had one; and two more, one on the 8th and one on the 10th of July.

"On the 10th of August, at 4 p.m., he had four fits, of a very severe nature, in quick succession, three occurring in less than five minutes and the fourth in about a quarter of an hour. When seen, he was in a state of profound coma; pupils contracted and insensible; pulse small, weak, and irregular; surface pale, with cold, clammy perspiration; breathing irregular, slow, and extremely laborious; the stertor was very marked; very little air seemed to pass into the lungs; the cheeks were puffed out during expiration. He was placed well over on his side, and propped up so that he remained about half way between the lateral and prone positions. There was an immediate flow of saliva from the mouth, and this was followed by a considerable quantity of sanio-mucous fluid. The breathing instantly became more free, and in less than a minute all stertor had ceased; the other symptoms gradually subsided, and in an hour's time, the bowels having been freely acted on by two drops of croton oil, there was a partial return of consciousness. The next morning he refused to stay in bed, and though dull and stupid, was moving about more or less all day."

Remarks.—The symptoms at the time were considered as rendering a fatal termination more than probable; whether such was really the case is immaterial. The sudden subsidence of the symptoms on the change of posture must, I think, indisputably prove that the cerebral congestion was relieved by it; and, further, we may conclude that the change in posture rendered a fatal termination *less probable* than if posture had not been attended to.

EXPLANATION OF PLATE.

The dissection from which the drawings were taken was made on the body of a woman, æt. 60, who died of cancer of the uterus. Great care was taken not to displace the parts more than was necessary to give a fair view of the base of the tongue and epiglottis. The sketches were taken with the subject lying on its back on the table.

Fig. 1. Showing the position of the tongue with the mouth closed.
Fig. 2. Showing the position of the tongue with the mouth open.

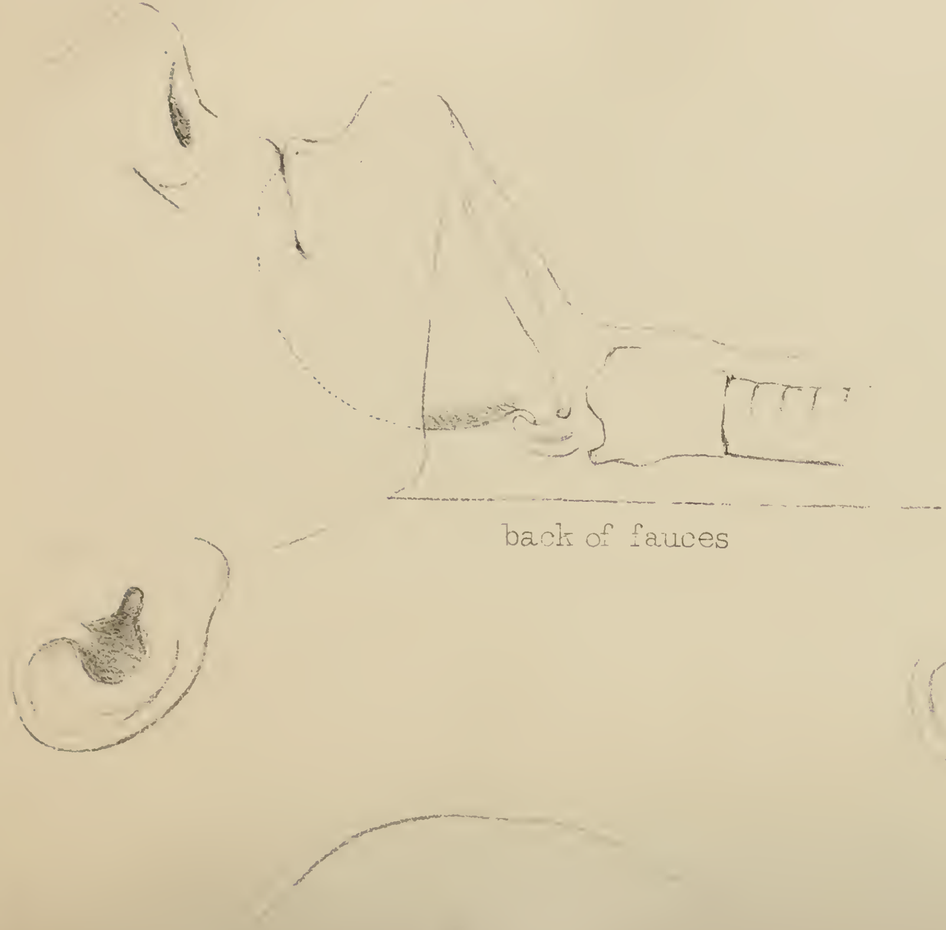
back of fauces

Fig. 1

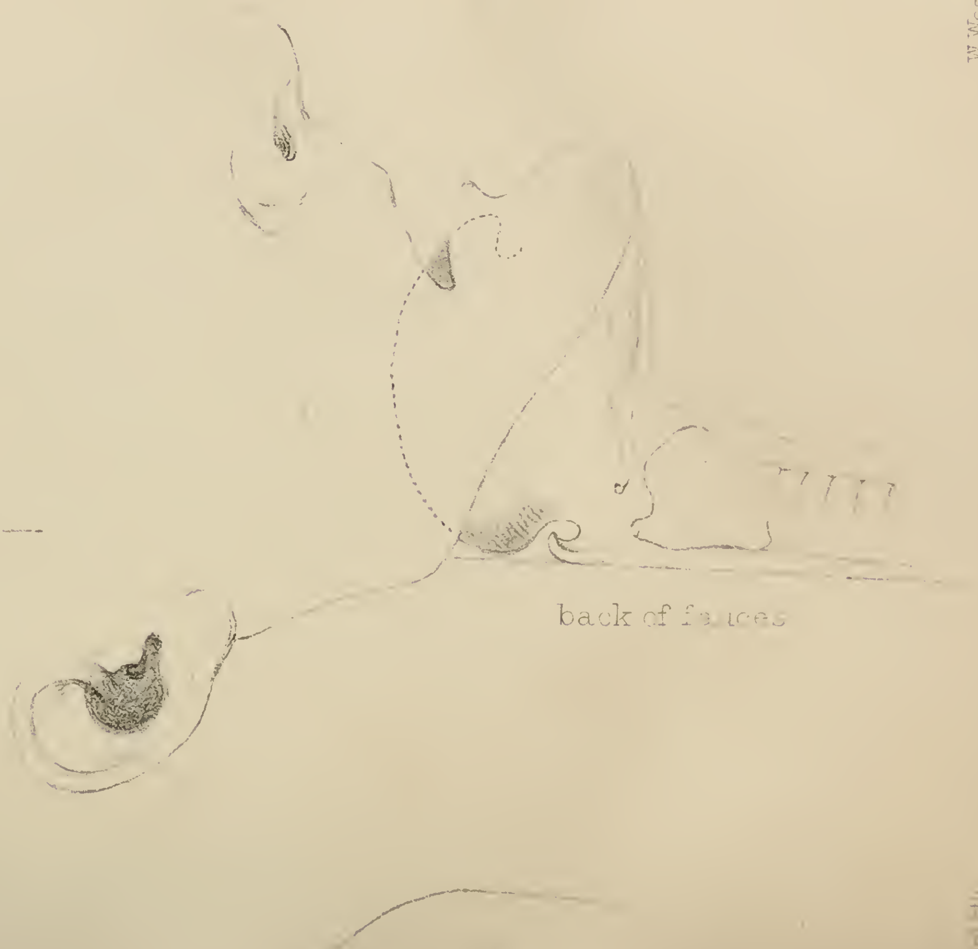
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Fig. 2

T. A. Ford lith.

W West imp

ON A FORM
OF
SECONDARY SYPHILITIC INOCULATION.

BY

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During the last session I had the honour of communicating to this Society an account of two distinct kinds of primary syphilitic inoculation. One of these forms was found to present the character of the adhesive, the other of the suppurative, inflammation. The former was shown not to be readily again inoculable on the patient who had the disease, while the latter was seen to be capable of being reproduced, either in the same or in any other individual, an indefinite number of times. The first was regarded as a disease which, in its natural course, infected the patient's system; the second as a local disease, requiring only local treatment.

It was also shown, in the communication referred to, that the specific adhesive inflammation, which did not ordinarily yield a discharge capable of being again inoculated upon the patient, might, under conditions of artificial irritation, furnish an inoculable secretion. The inoculations thus produced were, however, proved to be quite distinct from those which resulted from the inoculation of the discharge from naturally suppurating sores.

Secondary syphilitic inoculation has lately been regarded by a large number of writers as impossible, and experiments on a very large scale have been adduced in support of this opinion. But these experiments were made, for the most part, upon patients whose systems were already affected by the disease; and under these circumstances, as pointed out by myself in the year 1856, and as subsequently verified by others, the indurated chancre itself can seldom be inoculated again. It is not, therefore, surprising that the secondary effects of the same disease should, in like manner, not, as a rule, be inoculable upon those patients whose constitutions are already affected. If experiments are to be appealed to, they should be made upon those who have not previously had the disease; and as such experiments, for obvious reasons, cannot with propriety be tried, clinical observation must be relied upon for the general elucidation of this subject.

The following cases, selected from among others, and noted with much care, show that secondary syphilitic disease may be communicated by contact from one individual to another, and that one form in which it is so communicated bears a very strong resemblance to the primary indurated chancre. It is obvious, however, that secondary syphilis is not readily communicated by contact. Husbands and wives, children and their nurses, are constantly seen living together, where one party alone is affected, and the disease is not communicated to the other.

Many secondary syphilitic affections do not yield any fluid secretion, and this is probably a necessary condition for inoculation. In their ordinary state, these affections would not be inoculable, but clinical observation renders it highly probable that, under conditions of increased action, they may become the means of communicating the syphilitic disease. An indurated chancre which has ceased to be communicable to another part of the patient who bears it may, under irritation, be made to produce a kind of inoculation;[1] and secondary syphilitic affections, which are not capable of being communicated, under ordinary circumstances, to another individual, may, in like manner, under increased activity, become infectious.

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[1] 'Medico-Chirurgical Transactions,' vol. xlii, p. 438.

Case 1.—Frances H— was married in August, 1858. In January and February, 1859, she was under my care for sore throat, tubercles around the anus, and stricture of the rectum. These symptoms were supposed to be of a syphilitic nature, and the patient was accordingly subjected to mercurial treatment, which, however, she was unable to continue as long as was desirable.

In the beginning of May, the husband, who, up to this period, had not had syphilis, discovered a small red pimple on the prepuce. This pimple gradually enlarged, and in about a month assumed the appearance of a tubercle imbedded in the skin, without ulceration. A well-defined, cartilaginous induration, which terminated abruptly, gradually extended itself from this point, and became divided into two parts by a fissure caused by the retraction of the prepuce. With the exception of this fissure, there was, during the course of the disease, no ulceration nor abrasion of the surface. The accompanying drawing (see Plate I), by Dr. Westmacott, represents the induration as it existed on the 24th of September, and it also represents the glands in the groin, enlarged, but not inflamed. The condition of the inguinal glands in this case was exactly the same as that which is so well known as accompanying the primary infecting sore.

After the induration had fully developed itself on the husband's prepuce, the wife was carefully examined, and the edge of an abrasion or ulceration within the *os uteri* was distinctly seen. On the 23d of September, at her full period, she was delivered of a child, which, from its condition, was supposed to have died some ten days previously. About this time the husband became much emaciated, the posterior cervical glands enlarged, and some faint, copper-coloured blotches appeared on the forehead. The secondary eruption would, no doubt, have been much more developed, had it not been for the mercurial action to which he was subjected.

If testimony, obtained with much care, and under very favorable opportunities, is to be believed, neither husband nor wife had exposed themselves to contagion from any illicit source between the period of their marriage and the commencement of the disease in the man.

In this case, then, we have the wife infected with secondary syphilis before marriage, but living with her husband for nine months without communicating to him any disease. In the beginning of the year 1859 the uterus is called into increased activity; an abrasion or ulceration (which from the history of the case must be regarded as of a secondary syphilitic character) takes place within the os uteri, and then the husband presents a disease with all the characters of the specific adhesive inflammation.

Case 2.—Susan B—, æt. 66, presented herself at King's College Hospital on the 24th of October, 1853, with a scaly, copper-coloured eruption raised above the surface of the skin, and having in some parts a tubercular appearance. She had also a well-defined and extensive, indurated sore on the lip. She stated that she had never had any venereal affection, and had given birth to twelve healthy children.

Eighteen months before applying to the hospital she had noticed a pimple on the inside of the upper lip, which contained a little clear fluid. This broke, and a small sore formed, which, however, again healed in about a month, leaving a circumscribed induration. Three weeks ago the sore again broke out, and gradually extended until the date of her application at the hospital. An eruption made its appearance between three and four months from the first commencement of the pimple on the lip, and this had continued to recur at intervals ever since.

This old lady had taken her granddaughter to nurse, and having become exceedingly fond of it, was in the habit of constantly kissing it. Her son, she knew, had suffered from some venereal disease five months previous to the birth of the child; her daughter-in-law had died a few days after her confinement, and had not suckled her infant, which was consequently brought up by hand. When the child was ten weeks old some sores appeared on its tongue and lips. These were followed, a week afterwards, by an eruption on the nates, which was still visible when the grandmother applied at the hospital, and was clearly of a syphilitic nature. This woman might have, and probably often did, kiss her son with perfect impunity, but the syphilitic poison appears to have acquired increased activity with the new life of the child, and with that increased activity to have become more readily communicable by contact.

Case 3.—Mr. and Mrs. — were married in August, 1854. Mr. — had contracted syphilis in August, 1853. The sore remained open for three months. About a month after it had healed, a secondary eruption appeared upon the skin. Four months before his marriage he considered himself quite well; there were the remains of a syphilitic eruption, however, visible, and on the inner fold of the prepuce a very small, circumscribed induration could still be felt. A month after marriage, Mrs. — perceived an indurated spot on the left labium, which was sore when touched; some small tubercles subsequently formed, and the glands in the groin became enlarged, but not inflamed.

In the beginning of December this patient had some condylomatous excrescences at the margin of the anus, and an extremely painful fissure of the rectum. In another week an unmistakeable syphilitic eruption covered the body. She had not up to this time become pregnant. In this case, again, simple contact would not have been sufficient to communicate the disease; but under increased action the poison seems to have gained an activity which it had not before, and to have become again contagious.

It has been formally and perseveringly denied that secondary syphilitic disease can be communicated by contact from one individual to another. A theory has been based upon such assertions, which, in the name of science, has inflicted much domestic misery, and has often been the cause of the most painful and unjust accusations. There is but too much reason to believe that husbands have not unfrequently been led to suspect the fidelity of their wives, when they themselves were alone to blame;[1] and, on the other hand, men have been most unfairly accused of having contracted fresh syphilitic disease after marriage, when in reality the symptoms have arisen from the recurrence of an old disease of which they may have thought themselves perfectly free.

Case 4.—A gentleman and lady were married in the year 1859. Two months afterwards the wife experienced some irritation about the labia, which was followed by specific enlargement of the inguinal glands on both sides. Four months later there was a well-marked syphilitic eruption on the body, and enlargement of the posterior cervical glands. There was also at this time the remains of an indurated tubercle, which did not appear to have ulcerated, on the left labium, and the inguinal glands remained enlarged, but not tender nor inflamed. This patient had not become pregnant; the husband had very slight remains of an eruption, the result of syphilis contracted four years previously. There was also very slight remaining induration in the site of the original disease, and the skin in the immediate neighbourhood had since his marriage been occasionally inflamed and excoriated. In this case the friends of the lady, having received imperfect medical information, accused the husband of having contracted fresh disease after his marriage.

The form of secondary syphilitic inoculation to which I have now drawn attention commences by a chronic adhesive form of inflammation, which terminates in a perfectly circumscribed thickening. This may be raised from the surface in the form of a pimple or tubercle, or it may produce an induration not at all raised above the surrounding parts. The cuticle is generally abraded over the affected surface, but ulceration is no essential part of the disease. These secondary inoculations frequently indeed do ulcerate, but they also often run their course without any material loss of substance. When ulceration does occur, it not unfrequently manifests itself at the time when secondary or constitutional symptoms are about to appear. A period of incubation, varying from one to six or seven weeks, generally occurs between the contagion and the manifestation of the symptoms; any idea of destroying the poison with caustic within the first few days is here quite out of the question. Even if applied immediately upon the disease being perceived, it will not check the morbid action. This may have taken some four or five weeks to develope itself, and cannot now be counteracted by the destruction of a small portion of the tissues which have imbibed the poison. Excision of the indurated part will not stay the disease; the cut surface will take on the specific morbid action. Having, however, on several occasions removed specific indurations, I am inclined to think that the patients have ultimately done much better than they would have done had the original condition been allowed to remain. The subsequent indurations which have taken place on the cut surfaces have appeared to be more under control than those which first form in the course of the natural disease.

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[1] The number of instances in which unmarried women have been unjustly accused, and had a twofold injury inflicted upon them, by those under whose protection they are said to have been, is probably very much greater.

In considering the manner in which the venereal poison was communicated to the inhabitants of the South Sea Islands, Hunter observes that it was almost impossible to carry a chancre so long a voyage. Any difficulty there might be upon this head immediately disappears, as soon as the contagious properties of secondary syphilis (under certain circumstances) are recognised.

Babington had affirmed long ago that the induration might precede the ulceration[1] in syphilitic sores. This is formally denied by Ricord.[1] In the form of secondary syphilitic inoculation under consideration, the induration undoubtedly does appear when there is a slight epithelial abrasion only upon the surface, and may exist for weeks without any action that can be designated as ulceration. This character, however, does not serve to distinguish the effects of the secondary from those of the primary syphilitic inoculation. In both the induration is the most common characteristic of the disease; in both the ulceration may be entirely absent.

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[1] 'Works of Hunter,' by Palmer, vol. ii, p. 321.

Case 5.—In the beginning of November a patient came under my notice at King's College Hospital, with a well-marked, circular induration, terminating abruptly in the upper part of the prepuce. He had also a viscid discharge from the urethra.

During the whole of the month of November the induration gradually increased; the epithelium was abraded from a circular patch over its centre, and a whitish fluid was secreted from its surface. Beyond this there was not, during the whole of the period, any appearance whatever of ulceration. Upon microscopical examination, the secretion was found to consist of granular matter and epithelial débris, floating in a transparent fluid. It contained no pus-globules. The accompanying drawing (see Plate II) represents the induration on the prepuce, and a corresponding red patch on the glans penis. There was no induration in the latter situation.

Case 6.—The wife of the subject of the last case was admitted into the hospital, and carefully examined. An extensive induration was found on the opposed surfaces of the labia, and extending over the inferior commissure. The parts were red, tender to the touch, and denuded of epithelium. They secreted a whitish fluid, which, upon examination by the microscope, was found to contain granular matter and epithelial scales, but no pus. This patient had been out of health four months, having exposed herself to contagion six months before her admission into the hospital. Three months before her admission she felt a burning pain, with itching, and swelling of the labia, accompanied by slight discharge, and pain on walking. The inguinal glands also became specifically enlarged.

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[1] 'Leçons sur le chancre,' p. 88.

As these two last-mentioned patients had constantly lived together for twelve months, and as neither of them had previously had any syphilitic affection, it appears evident that the non-ulcerating disease communicated to the husband was of a primary nature; and that there was a period of incubation of considerable length before the disease manifested itself. Neither the character of the induration, nor the period of incubation, then, will suffice to distinguish the primary from the secondary form of natural syphilitic inoculation; and in the absence of a correct history, we have, I believe, no means of saying positively, in any individual case, whether the poison was derived from a primary or a secondary affection.

The proportion of indurated sores upon the face and head is much larger than in any other situation. This probably arises in part from the face and head being exposed to secondary syphilitic inoculation in a much greater relative degree than other parts. The same principle may account for the fact that syphilitic sores on the fingers are generally of the indurated kind; but here the proportion approaches much nearer that in which other parts of the body are affected.

The following cases may be regarded as probable instances of natural secondary syphilitic inoculation, although, as shown in the preceding remarks, the same form of disease may be communicated either by primary or by secondary syphilis.

Case 7.—A gentleman, who had been married several years, unfortunately exposed himself to contagion on the 24th of March, 1856. On the 2d of April, when I first saw him, there was a circumscribed abrasion on the upper and back part of the glans penis. This presented exactly the appearance as if a limited portion of cuticle had been peeled off, leaving a red, smooth, and glazed surface. Knowing, from previous experience, that sores in this particular locality do not become indurated as they do elsewhere, my attention was particularly directed to this point. The patient attended regularly, and at every visit I examined carefully the condition of the parts. From first to last no induration could be perceived around the sore. On the 29th of April a syphilitic eruption had made its appearance on the skin; and now, for the first time, a well-marked and circumscribed induration of the prepuce, in a part not previously affected, was perceived. This induration never evinced the slightest disposition to pass into ulceration. The patient had never before had syphilis, and recovered under the use of mercury.

As the induration may exist in the specific adhesive inflammation without the ulceration, so may the ulceration or abrasion, as proved by the preceding case, exist for a time at least without the induration. They may follow each other at an uncertain interval upon the same spot, or they may, in a few rare cases, such as the foregoing, be separated, and each condition incident to the disease may appear at a different part.

The period of incubation, which has so frequently been said never to exist in any form of syphilitic inoculation, was well marked in the following cases.

Case 8.—A first lieutenant in Her Majesty's navy, after having been five weeks at sea, saw a small pimple on the prepuce, which became abraded or ulcerated. He was told that it was nothing. The disease, however, persisted, and he was then subjected to a course of mercury whilst at sea. A secondary eruption followed, for which he underwent a course of calomel baths in the autumn of 1859.

Case 9.—A young midshipman, on his return from sea, had intercourse with one woman only. He had a viscid discharge from the urethra, for which he attended very regularly, as he was most anxious to get well before he again went to sea. In a month the discharge from the urethra suddenly ceased, but an induration, accurately circumscribed, and accompanied by a superficial abrasion, appeared immediately behind the glans penis on the right side. The corresponding inguinal glands soon became enlarged and hard, but not inflamed. The induration slowly increased, and there was every reason to suppose that it would soon be followed by a secondary eruption, when the further observation of the case was prevented by the patient being obliged to join his ship.

Case 10.—A gentleman had a superficial abrasion on the right side of the prepuce, for which he sought medical advice as soon as it appeared. It yielded very little secretion, and soon became surrounded by specific induration. The secretion was frequently examined by the microscope, but never, as far as was ascertained, contained any pus. An inguinal gland on the right side became enlarged, indurated, but not inflamed, and a secondary eruption followed. From peculiar circumstances, this gentleman was enabled to fix the period between exposure and the appearance of the abrasion at exactly one month.

Case 11.—A young lad presented himself at King's College Hospital on the 2d of December, 1859, with a well-marked circular and circumscribed induration in the upper part of the prepuce, immediately behind the glans. He had had one connexion only; this was followed by a discharge from the urethra, unaccompanied by any ardor urinæ. After the lapse of a month, the discharge ceased, and the induration made its appearance; this was at first accompanied by abrasion of the surface, which, however, soon again became covered with cuticle exactly resembling that of the surrounding parts. The glands in the groin were specifically enlarged.

Case 12.—A gentleman, whose wife was ill, had one intercourse only with a woman he accidentally met. Some time after, an abrasion appeared on the prepuce, for which he got the best medical advice he could obtain in Dublin and elsewhere. He was told that his disease was of no consequence. A month after exposure to contagion, the disease altered its character, and soon presented a circumscribed induration, slightly ulcerated upon its surface. The induration was excised, and the patient was put upon a course of calomel baths. The cut edges became indurated, and a slight eruption on the arm followed. But for the previous history, this eruption would scarcely have been suspected to have been syphilitic. Fifteen months afterwards this patient was perfectly well, having had no recurrence of any disease.

A very considerable number of cases have now fallen under my observation in which patients have been told by surgeons of eminence that their disease was of no consequence, and where the slight abrasion or ulceration which they may have had has subsequently become specifically indurated, and has been followed by secondary symptoms, often of the most troublesome kind. There is no doubt that medical men have often been misled by trusting to the assertions so often and so unreservedly made, that the specific characters of a chancre always follow immediately upon inoculation; and that the induration in the infecting variety always shows itself within the first few days.

The preceding cases are sufficient to show that, in one variety, at least, of infecting disease, a period of incubation does exist, both with regard to the induration, and also with respect to the ulceration or abrasion. The former, as has been stated, may appear without the latter, and the latter may be present for a time without the former. The absence of induration during the earlier periods of the disease is, therefore, no safe criterion as to its non-infecting[1] qualities. A discharge from the urethra not unfrequently precedes the form of disease now under consideration. This discharge may easily be mistaken for gonorrhœa. It differs, however, in being more viscid and tenacious in its nature; in not being accompanied by the same amount of irritation or ardor urinæ; in its short duration, and often abrupt termination. It is quite possible that it was this kind of matter with which Hunter inoculated himself when he produced a thickened sore, that was followed by secondary symptoms.[1]

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[1] Infection is used to indicate the reception of the poison into a patient's system; contagion, the transmission of the poison by contact from one individual to another, or from one part to another.

The disease which I have attempted to describe has, then, a period of incubation of about a month's duration, but varying, nevertheless, in certain cases, from six or seven days to as many weeks. It may be preceded by a discharge from the urethra, which generally ceases upon the appearance of the specific disease. The first symptom of this specific disease may be a slight ulceration; or it may be an induration, with a superficial abrasion of the epithelium only, unaccompanied by any ulceration; or the ulceration and induration may appear simultaneously. The disease is accompanied by specific indolent enlargement of the inguinal glands, and is followed, if left to itself, by constitutional syphilis.

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[1] The discharge in question does not appear in any way connected with ulcers within the urethra.

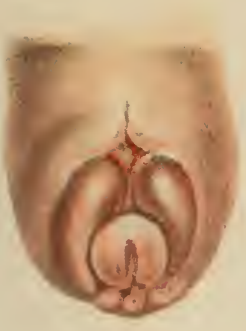
DESCRIPTION OF PLATE III.

I.—(Described as Plate I in Mr. Lee's paper), showing secondary non-ulcerating syphilitic inoculation.

I*a*.—Enlarged, but not inflamed, inguinal glands, accompanying the above-mentioned inoculation.

II.—(Described as Plate II in Mr. Lee's paper), representing primary non-ulcerating syphilitic inoculation.

1



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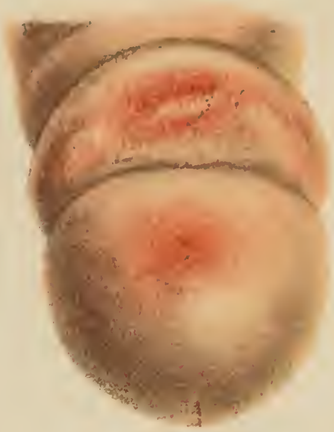

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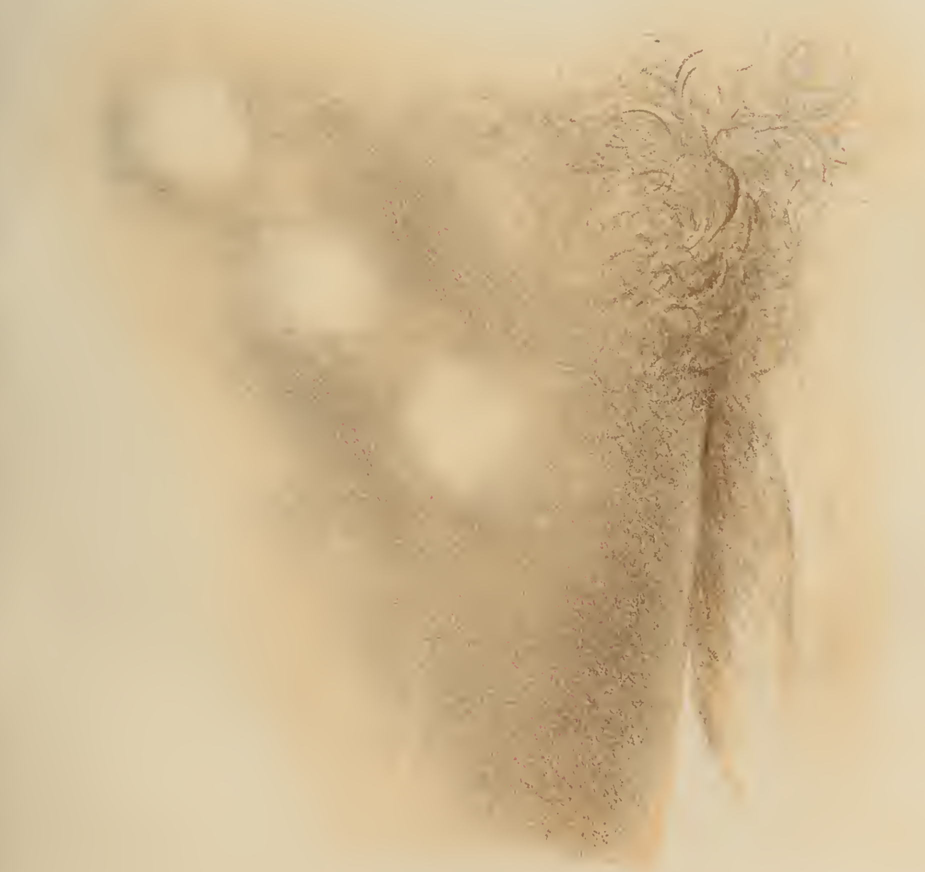

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ON

A NEW METHOD

OF

OPERATING FOR THE RADICAL CURE OF HERNIA.

BY

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The operations that have been hitherto devised for the cure of hernia may be classed under three heads, viz.—1st. Those which deal with the interior of the sac only, with a view of causing adhesion of its surfaces, either by a seton passed through it, as in Ragg's method, or by the injection of a solution of iodine or some irritating fluid, as practised in America and elsewhere. Without considering the frequent occurrence of peritonitis after the latter plan has been adopted, both are open to the objection that, even supposing that they are effectual in causing adhesion of the sides of the sac, the abdominal openings and the inguinal canal are left as patulous as before; while the loose connexion of the peritoneum with the fascia transversalis, in this situation, permits it readily to furnish another sac for a fresh protrusion. 2d. That of pinching up the anterior wall of the inguinal protrusion by a needle passed across it through the skin, with the object of causing adhesion of the opposed serous surfaces, as proposed by Bonnet, of Lyons. This method evidently deals only with that part of the hernia which protrudes through the external abdominal ring, leaving the upper part of the sac in the inguinal canal untouched and with a constant tendency to push down. 3d. The method of invagination of the skin into the canal. Upon this principle are founded two operations, that proposed by Gerdy, and the later one practised by Wurzer and Rothemunde.

In Gerdy's method, the skin and fascia covering the sac are invaginated upon the finger of the operator, and held in that position by a ligature thrust through the intercolumnar fascia and skin of the groin, till adhesion takes place at the point of ligature. An endeavour is also made to produce adhesion of the interior surface of the invaginated skin by the use of caustics and ligatures at the lower opening of the invagination; but this, I believe, was a failure. This method evidently deals only with the anterior wall of the hernia, and with the anterior fold of reduplication of the hernial sac. To make this intelligible, I must refer to Pl. IV, fig. 1, in which it will be seen that the reduplication of the sac leaves a posterior fold (*s*), which, on account of the closer attachment of the sac to the cord in that part, is usually longer, and descends lower than the anterior. The ligature evidently can only produce adhesion of the anterior fold, and other anterior structures, at *a*.

The method proposed by Wurzer substitutes for the finger of the operator a wooden plug, variously modified with the intent to fill the canal and openings, and to stretch them so much as to set up adhesive inflammation all round the invaginated sac. This plug is held in its position by one or two needles passing through the anterior wall, and connected with an external compress, intended to squeeze the anterior fold between it and the plug, so as to produce adhesion of the serous surfaces in its whole length. The essential difference between this method and Gerdy's consists in the retention of the plug till it has set up adhesive inflammation by its dilating pressure upon the opposed surfaces of the sac and the boundaries of the canal; the theory being that this will take place to such an extent as to prevent the unfolding of the invagination, which is thus supposed to plug up the openings by an inverted hollow cone of skin and fascia, as the neck of a bottle by the cork. The attempt is also sometimes made to cause the sides of the inverted cone to adhere by caustics and pressure.

Now, since in all these methods of producing a radical cure, the sac of the hernia is intended to be punctured and caused to inflame sufficiently to produce adhesion, the danger of peritonitis, which is regarded by many as a serious objection to any operation of this kind, may be considered as pretty nearly equal in all. From this, however, we must except the practice of injecting irritating fluids, which are obviously very likely to pass into the peritoneal cavity, and have often in this way proved fatal. But the results of the numerous cases operated on by the other methods that have been recorded, especially by Wurzer's, seem to prove that the danger of a fatal result is by no means great, nor sufficient to deter the surgeon from endeavouring to perfect our means of cure of this common deformity. In the method I have followed it will be seen, that it is by no means essential in recent and small cases of hernia to puncture the sac, since the most important manipulations can be effected altogether external to it, the sac being separated from its slight attachments and pushed up before the finger; and this, when it can be done, very much diminishes even the small chance there remains of peritoneal inflammation. A much more awkward objection is drawn from the inefficacy of these methods. Without wishing in any way to disparage the results of the operations I have thus briefly alluded to, or to magnify my own plan at the expense of others, I may say that in the hands of those surgeons from whom I have been able to obtain an account of the results of Wurzer's (the last and most extensively employed) and in all those cases which have come under my own observation, it has been entirely unsatisfactory; the rupture re-descending either immediately on leaving off the instrument and assuming the erect posture, or after discontinuing the truss worn after the operation. And this, I believe, is a very general impression among the surgeons in this country, as well as in France and Germany. In some cases I have heard of, it has rendered the wearing of a truss afterwards painful, if not impossible.

The causes of failure appear to me to be the following:

1st. The chief reason of the recurrence of the hernia, both in Gerdy's and Wurzer's operations, is the inefficiency of the steps taken to cause adhesion of the surfaces of the posterior fold of the invaginated sac together, and to the posterior wall of the hernial canal. Into this fold, forming thus a secondary sac, the descent of a knuckle of intestine or omentum is imminent, and its dilatation, and the consequent unfolding of the adherent portion in front, is only a question of time. In Gerdy's method no attempt is made for the closure of the posterior fold. In Wurzer's, the dilating pressure of the plug is supposed to produce this adhesion. To this theory the yielding nature of the structures, and the impossibility of producing a necessary amount of dilatation and pressure, without unbearable pain, offer the first objection.

2d. The intervention of the skin and fascia between the plug, the sac, and the parietes of the canal in which it is intended to produce adhesion. These are structures intended by nature to protect the subjacent parts invested by them from the effects of pressure and injury. Here we have the exertions of nature against those of the surgeon, and generally, I believe nature to have the best of it. It is said, moreover, that those cases make the most permanent cures in which sloughing of the skin and fascia at the apex of the plug takes place. This result, though effectual in overcoming the resistance of the tissues, makes the operation in these cases a very severe and clumsy one, and the recovery long delayed.

3d. The action of the plug is to dilate the openings and the canal, instead of contracting them, and thus they are rendered more ready to admit hernial protrusions than before. The principle of plugging up in this manner a dilatable opening with the idea of closing it, is surely a false one. In the cases I have examined after Wurzer's operation has been performed, the external ring and the canal have been remarkably patulous.

4th. The elastic reaction of the skin, and the weight of the testis and scrotum, are enabled by this patulous condition of the external ring, to drag down the invaginated tissues, by exerting their force upon the adhesions at the upper part of the canal, and thus aiding in the dilatation of the posterior fold. The hollow cone of skin is usually totally obliterated by this process in a greater or less interval of time after the operation.

From what I have seen in the treatment of these cases, I believe that much of the mass of inflammatory thickening and exudation obtained by artificial means, is temporary only; the parts sooner or later assuming their normal flexibility and mobility. But if by means of the adhesive process a close approximation of the normal and permanent transplanted structures is obtained, these afford in themselves the best obstacle to the descent of the bowel. The absorption of the mass of exudation is, I am convinced, accelerated by the constant wearing of a truss soon after the operation.

5th. In many cases with narrow canals, failure is owing to the impossibility of invaginating the skin as far up as the internal opening, a mere indentation only being accomplished, leaving at the top of the canal a portion of sac ready for the formation of a fresh hernia. In some cases I have operated on, I have found the sac much narrowed at the external ring, and widening considerably towards the inner opening, so as to be funnel shaped. These obviously would not admit a sufficiently large plug to act on the internal opening, except by a preliminary process of dilatation of the external ring, which seems to be a painful process, and one objectionable in principle. The dilating plug used by Mr. Davies, of Birmingham, is intended to meet these cases, but does so, I have no doubt, at the expense of much pain to the patient and sloughing of the skin.

Now, in order to avoid these sources of failure in operating for the radical cure of hernia, it seemed to me better to proceed upon a principle directly opposite to that of dilatation employed in Wurzer's, namely, that of drawing together and compressing by ligature the abdominal opening and the inguinal canal, to cause to cohere closely together, by adhesion in the track of the encircling ligature, the structures which form their sides and boundaries, and to agglomerate a solid mass, not of new and adventitious tissue, but of the more permanent tendinous and fascial structures. And to accomplish this more effectually, both by obtaining a higher point within the canal, and a more distinct perception of the anatomy of the parts operated upon, and by lessening as much as possible, and at the same time rendering as effective and permanent as possible, the interposed and transplanted tissue, it seemed better to give the operation a subcutaneous character. These two principles combined in the use of a subcutaneous ligature are, so far as I am aware, new in their application to the radical cure of hernia, and, indeed, I am not aware that they have been employed in this combination by other operators in any other surgical proceeding. I relied the more on their truth, in that I had been successful on many previous occasions, in the treatment of subcutaneous nævi on the face and other exposed situations, without producing a loss of skin or much scar, by the application of a "subcutaneous clovehitch," which was followed by a remarkable contraction and consolidation of the subcutaneous structures. One of these cases was published in the 'Medical Times and Gazette' some months ago.

Description of the operation.—The pubis and scrotum of the affected side being shaved, the patient should be laid upon his back with the shoulders raised. The surgeon will find it most convenient to stand on the side to be operated on. The hernia must then be reduced, and held up by an assistant pressing with his fingers over the internal ring. With a small, narrow-bladed tenotomy knife, an incision is made to the depth of the skin only, of about three quarters of an inch in length, in the scrotum, over the centre of the lower part of the hernial tumour, at least two inches below the spine of the pubes. (Diagram IV, fig. 5.) Then the knife, being insinuated flatways between the skin and fascia, is made to separate the former from the latter all round the edges of the incision, over an area of at least two inches in vertical, by one and a half inch in transverse, diameter. This, from the loose attachment of the skin at this part, is easily done. The knees of the patient should next be drawn up towards the abdomen, and held together, so as to relax the structures connected with Poupart's ligament. Then the forefinger of the operator is passed through the opening in the skin, and made to invaginate the detached fascia through the external ring into the inguinal canal. This will be most effectually done by using the right hand for the right side, and *vice versâ*. The operator should endeavour to commence the invagination of the fascia from as low a point as the external incision will admit, so as to push the finger as much as possible between the sac of the hernia and the spermatic cord, which should, at the same time, be put on the stretch by traction upon the testis with the disengaged hand. (Pl. V, fig. 1.) The invaginating finger should be made to reach the internal opening of the hernia.

The position of the spermatic cord, lying above and behind Poupart's ligament, can then be distinctly made out. When the point of the finger has reached the internal opening, it will be placed behind the lower border of the internal oblique muscle, the position of which will be made more evident to the operator by hooking it forward upon the point of the finger. (Pl. V, fig. 2.) He will then be sensible of the edge of the conjoined tendon (into which the muscle is inserted internally) at the outer side of his finger (in reference to his own hand) passing downwards behind the canal, to be inserted with Gimbernat's ligament into the pectineal line of the pubis. Next, a stout, blunt-pointed needle mounted on a strong handle, with a well-marked curve (as seen in the figure), is to be passed along the outer border of the invaginating finger, and pushed through the conjoined tendon and internal pillar of the external ring, till the point is seen to raise the skin on the surface of the finger. In this manœuvre the point of the needle must be carefully preceded by that of the finger. The skin is then to be drawn inwards and a little upwards, as much as its attachments will allow it to slide, and the point of the needle pushed through. A strong, smooth, hempen thread (well waxed, and then soaped) is then passed through the needle's eye, and the latter withdrawn, leaving one end of the thread in the puncture. The invaginating finger is then passed behind Poupart's ligament, between it and the spermatic cord, as far from the edge of the external pillar of the external ring, and as near to the internal ring, as possible. The ligament may then be raised forwards towards the surface upon the finger. (Pl. V, fig. 3.) The needle carrying the end of the ligature, which it has before withdrawn, must then be passed along the internal border of the invaginating finger to the deep surface of Poupart's ligament, about its centre (midway between the iliac and pubic spines), and pushed carefully through towards the skin. When its point begins to raise the skin, the latter is to be again slid outwards and a little downwards, till the point of the needle appears at the puncture before made: through this the instrument is to be a second time pushed, the loop of the thread seized, and the needle again withdrawn, leaving a loop in the puncture, and carrying the free end of the thread. The invaginating finger is then to be again placed on the inner side of the spermatic cord, which is to be pushed outwards by it. The point of the finger is then to be pressed against the centre of the posterior wall, so as to push backward the conjoined tendon, &c. (Pl. V, fig. 4.) The thick, tendinous structure, formed by the triangular ligament and conjoined tendon, will then be felt covering the edge of the rectus muscle on the inner side of the finger, raised by the muscle considerably in relief. Into this the needle is to be next thrust, so as to take up a portion of it about half an inch above the pubic spine, close to its insertion into the pectineal line, and external to the border of the rectus muscle, which affords a very plain guide, and protects the peritoneum from injury.

By sliding the original puncture in the skin downwards to its utmost extent, the needle can easily be passed a third time through it. The needle is then freed from the ligature and withdrawn. We have then, passing through the puncture of the skin in the groin, the two ends of the ligature and a central loop. The upper of the ends passes through the upper part of the conjoined tendon, where the fibres of the oblique muscle begin to be inserted on it; the lower, through the triangular ligament and conjoined tendon, close to the insertion of Gimbernat's and Poupart's ligaments into the pubis; and both through the aponeurosis of the external oblique forming the inner pillar of the ring. The central loop passes through the centre of Poupart's ligament and the lower fibres of the internal oblique muscle arising from it. Thus, we leave in the canal two threads, passing across the invaginated fascia, and, in large and old cases, across the sac, immediately above and in front of and investing the spermatic cord, and connecting the inferior and external boundary of the canal, or Poupart's ligament, with the superior, internal, and posterior boundaries, formed by the fascia transversalis, conjoined tendon, and triangular ligament. (Pl. IV, fig. 2.) A pad of boxwood, or, what is still better, of glass or porcelain (*c*), about two inches long and one inch wide, flattened so as to be oval in section, and with the ends rounded off, is next applied obliquely upon the skin, along the course of the inguinal canal, with the centre opposite the puncture. The two free ends of the ligature are drawn to the outer side, and the loop to the inner side of the pad, so as to cross beneath it. One end of the thread is then passed through the loop and tied in a loop-knot to the other. The advantages of the double ligature are—1st, that of additional security against immediate protrusion; 2dly, that of producing action in the whole extent of the canal by obtaining two tracks and a broad intervening surface of adhesive effusion across the sac and fascia, one close to the internal ring, and the other below, near the external; and 3dly, the more effectual occlusion of the external ring, upon which the lower ligature directly acts. By keeping the two ligatures upon one thread, and making one knot suffice, we obtain a more equable adjustment of the pressure when the swelling supervenes. By tightening the string before the pad is placed, the surgeon may obtain direct evidence of the effect of the ligatures in closing the canal, on placing his finger therein. If the ligatures be properly placed, the canal will, even though a very wide one, be found completely occluded. The posterior wall should be felt drawn forward to meet the anterior. If this be not distinctly perceived, I consider the threads are not properly placed. I may here remark that the success of the operation mainly depends upon the effective closing of the canal by this drawing together of its anterior and posterior walls, and I have no doubt that many failures in this point will happen to the surgeon till he becomes accustomed to the manipulation of the parts. After the ligature is tied firmly over the compress, the lower end of the latter should reach nearly to the pubic crest, up to which point the wound in the scrotum will be tucked. Pledgets of lint may then be placed on each side of the compress, and a stout linen pad over all, secured by a spica bandage. The patient should be carried to bed, his shoulders raised, and a long bolster under his knees. If he has a bad night, and much pain in the groin, the removal of the linen pad and bandage will much relieve him. As their value ceases after the first twenty-four hours, and they may be injurious in retaining any discharge that may form, I have lately made it a rule to remove them the next day. Pressure is beneficial only so long as there is a chance of the deeper-seated parts becoming adherent by the first intention. In some cases, the discharge is very trifling, the lower opening healing at once. In most the discharge is greater, being at first serous and reddish, but soon becoming thick and purulent. In large cases, where the sac is punctured, the serous discharge is sometimes abundant, and evidently proceeds from the interior of the sac. In one of my cases the sac below the ligatures sloughed; and the discharges and sloughs escaped freely by the lower opening. The ligatures caused effectual adhesion of the neck of the sac, and thus prevented extension of mischief to the peritoneum. This patient made an excellent cure, and was, at the same time, relieved of a varicocele which coexisted with the hernia. In no case should the lower opening be stitched up, as it is very useful if there be much discharge. The introduction of a drainage tube I have, in one case, found beneficial. In small cases, or where sufficient action seems to be quickly set up, I remove the compress at the end of the third or fourth day; in more sluggish cases at the sixth or seventh day; leaving, however, the threads in the puncture to act as a seton in promoting the escape of the discharges, and at the same time prolonging the effusive process in their track, and in the canal and sac, for as long as may be deemed necessary. The amount of solid effusion may be estimated by the swelling and hardness felt at the external ring, in the canal, and in the track of the ligatures. If the inflammation run high, and the discharge be copious, a poultice or water-dressing may be applied after fomentations. In the latter stage of the cure, a sulphate of zinc lotion may be injected with benefit.

The results intended to be obtained by the operation just described may be briefly recapitulated as follows. I may with advantage place them in the order of the importance which I attach to them severally.

1st. The posterior and superior boundaries of the dilated canal are drawn forwards and downwards towards Poupart's ligament, and become united by inflammatory effusion, in the area of pressure exercised by the ligatures, to the anterior and inferior boundaries. By the use of the two ligatures, this takes place from the internal opening above, to the external ring below. The effect of this adhesion will be seen in Plate IV, by comparing figs. 1 and 2 with 3 and 4, to make the posterior wall act like the limb of a valve (*v*), excluding the bowel by closing against the anterior wall (*a*). This action is aided by the contraction of the cicatrized tissues, and increased by the subsequent downward traction of the testis and scrotum. In this way we have an assurance, that the older the cure and the more the pressure, the greater the mechanical resistance and security against the return of the protrusion. The spermatic cord is closely embraced by the contracting tissues in the groove behind Poupart's ligament, which protects it from undue pressure.

2dly. The consolidation and contraction of the tendinous boundaries of the canal, in the track of the ligatures, render them more capable of resisting pressure from within, and so increase the strength of the parts afterwards. The effects of these cords of adhesion will be best understood by reference to Plate IV, fig. 3. It will be seen that the cicatrix in the skin of the groin, as seen in a vertical section at *a*, will be drawn backwards by the contraction of the circular cord of effusion, towards the internal parts and peritoneum at *v* and *p*, compressing more and more closely the opposite walls at the upper and lower extremities of the inguinal canal. Before fully estimating the importance of this result, it must be noticed that the primary dilatation of the canal in the formation of oblique inguinal hernia, is chiefly produced by the yielding of the superior and posterior walls at the upper part of the canal; the aponeurotic tendon of the external oblique at first resisting the anterior protrusion, and then directing it downwards and inwards in the course of the canal towards the external ring. This produces, in many herniæ, and indeed in most which are still bubonoceles, an infundibular form, with the apex at the external ring. In most patients who have a tendency to hernial protrusion, a bulging is apparent at the internal ring, from a deficiency in the development of the lower fibres of the internal oblique, which cover that opening in front. In muscular subjects, this is rendered much more prominent during the contractions of the recti muscles, which, by virtue of their compressing action upon the bowels, draw backwards with them their sheath, formed by the conjoined tendon above the point of splitting of the internal oblique portion of it. In these subjects there exists a disproportion between the muscular contracting force of the recti and the tendinous and muscular resisting forces at the internal ring; and the upper part of the canal is opened, as it were, by the backward traction of the recti muscles upon the conjoined tendon, as well as by the yielding of the anterior wall.

This action, associated with deficient development of the lower part of the internal oblique, appears to me to be the main cause of inguinal herniæ in muscular subjects. It will be seen that the two internal ligature-tracks of adhesion directly counteract the effects of this action, by drawing the cicatrix of the skin and anterior wall of the canal backward with the conjoined tendon, as may be seen upon the patients cured by this operation, by a depression of the cicatrix during the action of the recti muscles. It will also be seen that Poupart's ligament is thus made to act as a "point d'appui" of the resisting forces. It seems certain that no operation, which leaves the upper part of the canal unaltered, can produce a permanent cure of oblique inguinal hernia. The opening or sac which is thereby left affords an admission to a wedge of bowel or omentum, and being acted upon by the whole power of the abdominal muscles, speedily enlarges at the expense of the loosely attached and abundant peritoneum opposite the part. The return of the hernia in such circumstances is only a question of time.

3dly. The invaginated and transplanted fascia adheres on its opposing raw surfaces by means of a cord-like connective tissue, which is formed in front of and is adherent to the spermatic cord: by similar fibrous tissue formed in the tracks of the ligatures the fascia is further held firmly within the canal, and connected with the skin at the upper cicatrix, as well as with the anterior and posterior walls. (Plate IV, fig. 3.) To obtain this result, as well as to gain a more distinct perception of the parts within the canal, is an object, I think, clearly worth an incision of a half or quarter of an inch long in the skin of the scrotum, and a separation of the fascia. Nor can I agree with the principle of invaginating the skin, and holding it in the canal by ligatures attempted to be passed through the pillars of the external ring. By passing the skin through the external ring, the borders of the ring are separated, and prevented from adhering together. Again, without the incision it is quite impossible to get a hold with the needle upon the conjoined tendon; and not only are the pillars of the external ring insecurely fixed, but the cord and other important structures are much endangered by the instrument.

4thly. The fresh adhesions of the scrotum to the pillars of the external ring, while the latter are held together by the ligature, contribute much to close it, to bind it to the cord, and to hold up the transplanted fascia in its new position in the canal. The closure or obliteration of the external ring by the lateral traction of the lower ligature drawing together the pillars, may be remarked as one of the original features of the operation, and as contributing much to its success. In small cases of direct inguinal hernia it may be even sufficient to produce a cure, if care be taken to secure a hold with the inner end of the ligature upon the triangular fascia (or ligament) forming the posterior wall at this point and covering the edge of the rectus muscle.

Lastly. In those cases where the sac is large, has become adherent to the cord, and cannot be pushed back into the peritoneal cavity, the punctures through the sac, and the passage of the ligature-threads across it in front of the cord, as well as the grip of the ligature around it, produce adhesion of its surfaces to each other and to the spermatic cord, over the whole extent of the invagination, and not merely in its anterior fold. At the same time it becomes firmly united in one mass with the walls of the canal which are tied up with it. In recent cases, where the protrusion was but small, or a "bubonocele," I believe the patient to have been cured by obtaining the first four results, without puncturing the sac at all, but only by pushing it up before the invaginating finger, and working with the needle altogether outside it.

In noticing the objections that have been made to the operation, since it was first brought before public notice in May, 1858, I must premise that they have mainly, if not entirely, been founded upon *primâ facie* and theoretical grounds, or upon the results of operations said to be according to my method, but for the execution of which the plan is in no wise responsible. First, as regards the danger of the plan. It has been urged that the peritoneum, the spermatic cord, the epigastric vessels, and even the iliac vessels and bowels, are endangered by the passage of the needle. But it will be seen, by an inspection of Plate V, that if the operation be properly and carefully done, the invaginating finger of the operator is placed so as to fill up the internal opening and keep out the bowel, and also to intervene between the spermatic cord, and the fascia transversalis covering the epigastric vessels and peritoneum, on the one hand, and the end of the needle at the point of puncture, on the other.

The fascia transversalis is a structure of much importance in this operation, on account of its thickness and density, and the way in which it covers and protects the iliac vessels and their branches, the circumflex iliac and epigastric. It can readily be felt, by its resistance to the finger pressing back towards the peritoneum. Of course, some familiarity with the parts is essential to the hand of the surgeon before doing this operation on the living subject. Before passing the needle through Poupart's ligament, that structure should be raised forward upon the finger and lifted, as it were, from the femoral vessels below and behind it, which may, at the same time, be pressed backward and outward by the thumb of the invaginating hand, placed on the skin below. (See Plate V, fig. 3.)

The needle used in this operation is much curved. The concavity of the curve should be kept carefully directed towards the surface, to which the point must necessarily tend if this precaution be adhered to, the needle kept steady, and carefully protected with the point of the invaginating finger. The point of the instrument should taper abruptly, and be well rounded on the convexity; and on no account should it have a cutting edge. It should be adapted rather for splitting the tissues, than for cutting its way through; and should therefore not be too sharp. If this be not attended to, the tendinous fibres of Poupart's ligament may be cut as by a tenotomy knife, instead of punctured; and so the hold of the ligature upon it be destroyed.

The best answer to any objection as to the danger of peritonitis from interfering with parts so near to the peritoneum, is to be drawn from the total results of my cases, fifteen in number, and all inguinal herniæ, in addition to the great number of cases in which Wurzer's plan, which interferes rather more with the peritoneum than mine, has been followed without bad results. The severity of an operation is best judged of by the duration of the after-treatment. In only one case was the patient detained in bed for more than a month. This was a case in which copious suppuration and burrowing of matter between the muscles of the abdomen supervened about the sixth day, with tympanitis, possibly associated with some parietal peritonitis. Of the rest of the cases, one was up on the ninth day, one on the eleventh, two on the twelfth, two on the fourteenth, four on the eighteenth; one three weeks after, another one month. All have done well as regards safety to life. In all, except two or three, the general symptoms were so slight as to excite little attention. These results can hardly be said to support the objection of severity or danger. Nor can the slight incision in the scrotum, which favours the downward escape of the discharge, be considered to make this operation more severe, or more likely to be followed by mischief, than Wurzer's or Gerdy's, in which the instrument also punctures the scrotum. I ought to mention that as little disturbance of the tissues as possible is desirable in the operation. Much roughness increases the chances of bad results, of abscesses and inordinate inflammatory action.

Of the efficacy of the operation, the best proof lies also in the results of the cases; and, as there is no proof like ocular proof, I have in attendance to-night, for the inspection of the Fellows, six cases in which a cure has been effected. One is the first case on which I operated, one year and eleven months ago. Another is the patient in whom the symptoms were most severe after the operation; the cure in whom has been so perfect, that from the time of the operation he has worn no truss for four months. I have also present the patient in whom the cure was one of the most speedy, and the symptoms the most slight; he having been out of bed on the twelfth day after the operation. In this patient the permanence of the cure has been tried eight months, and most severely tested, first by a bronchitis, which came on during the progress of the cure, and next by his having been employed without truss in the laborious work of harvesting and forking hay last autumn. Another of the cases present has been cured of a congenital hernia, which has been thought most difficult to cure. This patient was operated on last June (eight months ago), and has worn no truss since the operation, nor before, although he has been subject to heavy labour (piling shells) at Woolwich. A fourth case is here for inspection, in which no truss whatever has been worn before or since the operation, which was done eight months ago. This is also a very complete cure, the only trace of the deformity being a slight bulging opposite the internal ring (present also on the other side), from tenuity of the abdominal wall. The cough impulse is evidently arrested above the upper cicatrix.

The most likely cause of failure in this operation consists in the operator placing his ligatures too low, and closing the external ring only, whereby he fails to obtain a hold on the posterior wall of the canal. This, though effectual in closing the external opening, merely converts the hernia into a "bubonocele;" and, since the attachment of the oblique muscles to each other is not very intimate, the hernia may reach a considerable size, and even become strangulated by protruding between them, and without passing into the scrotum at all. It is then only a question of time as to its descent into the scrotum; the power of the abdominal muscles being so great, that when once a sac, however small, admits the fluid pressure of a piece of intestine, the complete dilatation of the rings and canal must take place sooner or later, unless a truss is worn. The only efficient closure is the valve-like approximation of the sides of the canal. One other cause of failure consists in the stitches not being placed far enough on the posterior wall, but so as to permit the intestine to slip down behind them. This I have seen happen in more than one instance. The threads should so be placed, that the opening is completely stitched up, so to speak.

In all the operations that have been hitherto proposed for the cure of hernia, it has been thought necessary to cause a truss to be worn for some months after the operation. In my first cases I followed the usual plan; but, thinking that the pressure of the truss rather promoted the absorption of the newly formed tissues, I have, in the last five or six cases, dispensed nearly altogether with its use. In four of the cases that are present this evening no truss has been worn. And though it was partly with a view of more thoroughly testing the efficacy of the cure, as well as of giving more tenacity to the adhesions, that I, at first, left off the constant use of the truss, I am at present disposed, in favorable cases, not to recommend the wearing of a truss after the operation. If, however, the abdominal parietes at the internal ring be weak and bulging after the operation, as is often the case, even when no hernia has shown itself, the use of a weak truss, with a flat pad, during violent exercise or hard work, is advisable. Trusses with strong springs are, I believe, most injurious; if worn constantly, they favour very much the absorption of the effusion within the canal, and have, in my experience, given rise to varicocele. The occasional and intermittent use of a weak instrument is, on the contrary, beneficial, by exciting contraction in the canal. In two cases, after a sufficient length of time had elapsed to test the reality of the cure, I recommended the occasional use of a weak truss with this view. The usual effect of a truss worn continuously after an operation for the radical cure is to mask its failure. Perhaps the operation has diminished somewhat the internal opening. The accustomed knuckle of bowel does not find its way so readily into the canal. The hernia does not descend until the removal of the truss permits the effect of the abdominal muscles upon it. For this reason I think it desirable to test the reality of the cure before the truss is put on; and only in cases with weak, bulging groins should the truss be habitually worn.

In only one of the cases has the omission of the truss clearly given rise to a relapse; and this was entirely from the folly of the patient, a sailor boy, who very imprudently, and from over-confidence, attempted to lift a hundredweight of coals, three weeks or a month after the operation, and without a truss. The consequence, as might have been anticipated, was a rupture of all the adhesions and a reproduction of the hernia. It ought to be said, however, that in this case one ligature only had been used, the patient was only in bed nine days, and the cause of the return of the hernia was a greater effort than had been sufficient for the production of the original hernia.

EXPLANATION OF PLATE IV.

Fig. 1. Diagrammatic section, showing the posterior fold left after invagination of the scrotum and sac in Gerdy's and Wurzer's methods of operating.

a. The anterior point of adhesion.
v. The posterior wall of the hernial canal.
s. The posterior fold of the sac or space in the canal into which the intestine re-descends.

Fig. 2. A similar section, after the author's method, showing the course, position, and effect of the ligature and compress upon the invaginated fascia and sac and walls of the canal.

c. The compress, with the loop and ends of the ligature previous to tying.
v and *p.* The upper and lower perforations of the posterior wall (conjoined tendon).

Fig. 3. Ditto, showing the adhesions of the above-named structures after the operation, the consolidation of the anterior and posterior walls, and the circular contraction in the track of the ligatures.

Fig. 4 illustrates the valve-like action of the posterior wall in preventing the descent of the intestine.

Fig. 5 illustrates the first step in the author's method of operating, viz., the puncture in the scrotum and the separation of the fascia from the skin by the subcutaneous knife.

Fig 1.

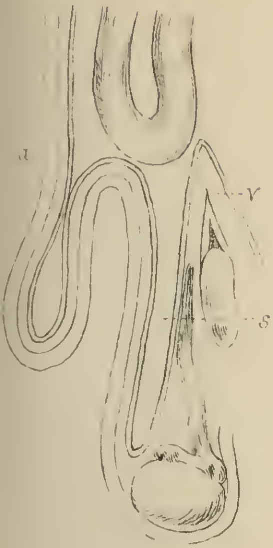


Fig 2.

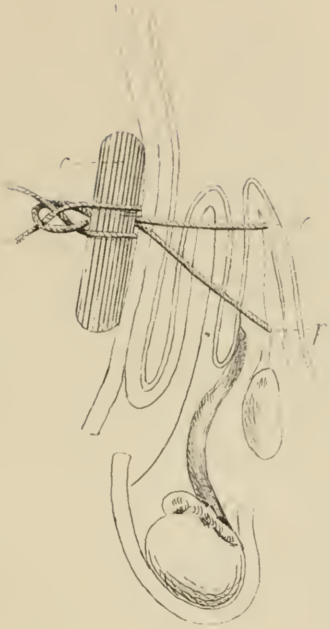


Fig 3.

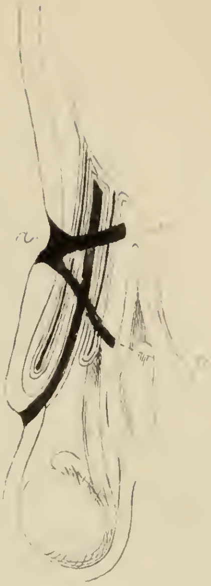


Fig 4.

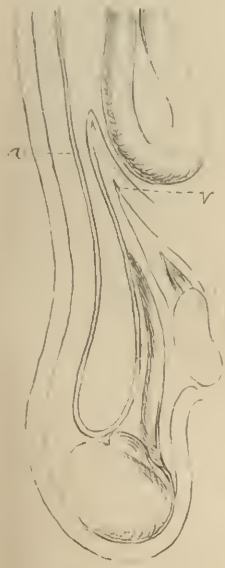
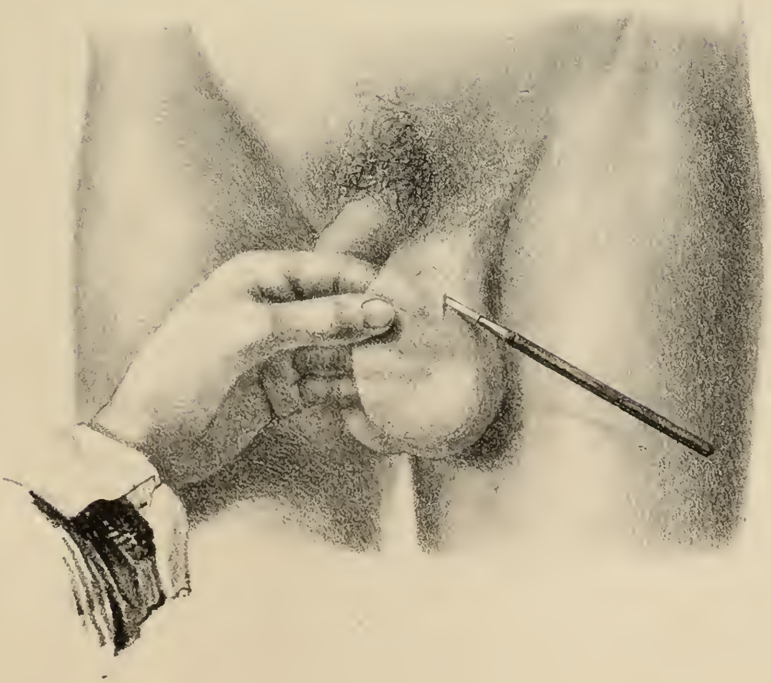


Fig 5.



H.Ford.

W.West imp

Fig 1.

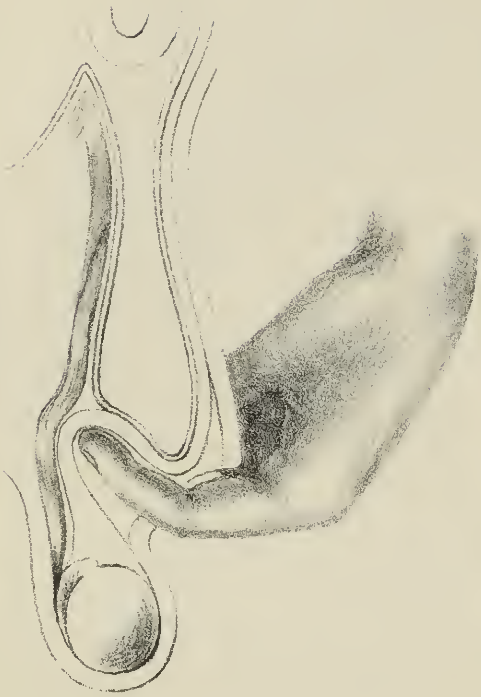


Fig. 2

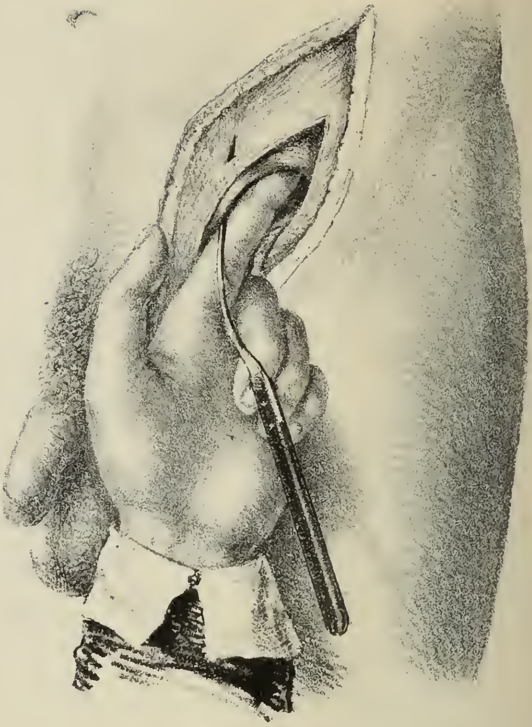


Fig 4.

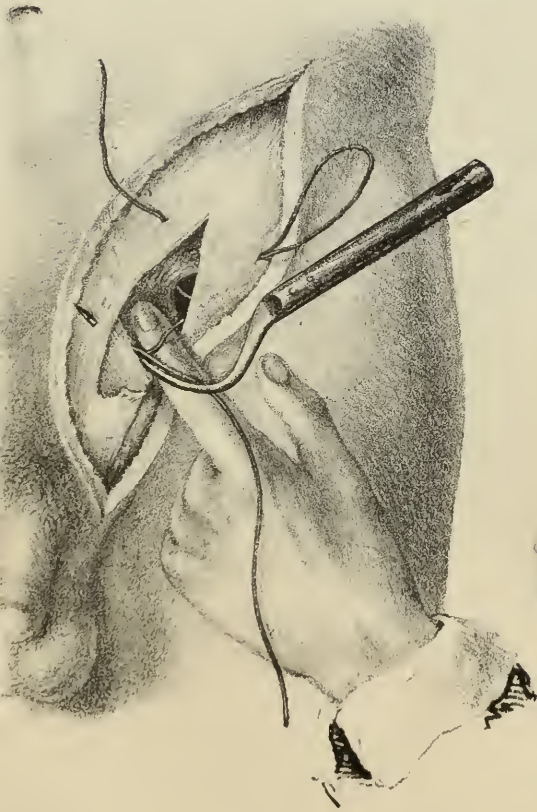
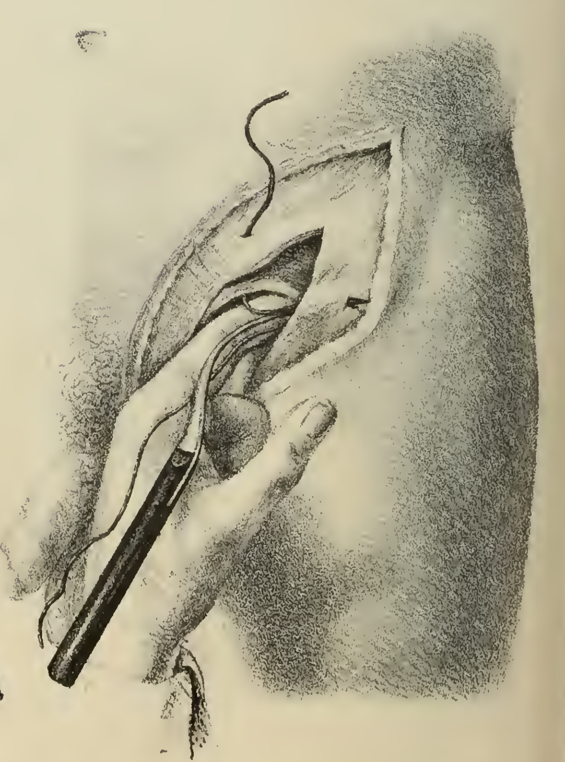


Fig 3.



H Ford

W West imp.

EXPLANATION OF PLATE V.

Fig. 1. Sectional drawing, illustrating the second step in the author's operation, viz., the passage of the forefinger through the skin of the scrotum, and the invagination of the fascia and sac.

Fig. 2 illustrates the third step in the operation, as exposed by a dissection of the integuments outwards. The aponeurosis of the external oblique is slit up in the site of the external ring, to show the forefinger of the operator raising the lower border of the internal oblique muscle, and the needle passing through the upper part of the conjoined tendon.

Fig. 3 shows the next proceeding. The end of the ligature is left in the first puncture; the surgeon's finger raises the external pillar directly above Poupart's ligament, and guards the point of the needle as it passes through, protecting the spermatic cord and deep vessels.

Fig. 4 shows the application of the lower end of the ligature to the triangular fascia, conjoined tendon, and internal pillar close to the pubis. The finger of the operator is seen to draw outwards the spermatic cord, and to protect it from the needle. The loop of the ligature is left in the second puncture, and the lower end of it through the last.

ON THE NATURE

OF

OVARIAN CYSTS

WHICH CONTAIN TEETH, HAIR, AND FATTY MATTER.

BY

ROBERT LEE, M.D., F.R.S., &c.

Received Dec. 19th, 1859.—Read March 13th, 1860.

Before the close of the last century, Schenkius, Schacher, Horstius, Le Rich, and Dr. Cleghorn had published cases in which ovarian cysts containing teeth, bone, hair, and fatty matter had been observed; and they had been deemed by them to be merely cases of extra-uterine or ovarian conception. Dr. Baillie was led to doubt the correctness of this view of the mode of formation of these cysts from the following case, which, he observes, "affords many reasons why we should be led to believe that the ovaria in women have some power within themselves of taking on a process which is an imitation of generation, without any previous connection with a male, and it is with this view that I proceed to relate it.

"In a female child, about twelve or thirteen years old, which was brought to Windmill Street for dissection, the right ovarium was converted into a substance, doughy to the touch, and about the size of a large hen's-egg.

"On cutting into the substance, I found an apparently fatty mass, intermixed with hair and an excrescence of bones. This circumstance startled me very much, as I had always been led to believe that such appearances were a sort of imperfect conception. The case being very singular, I was led to pay considerable attention to the change in the ovarium.

"The fatty mass was of a yellowish-white colour—in some places whiter than in others; it was very unctuous to the feeling, and consisted of shortened or separated particles, not having the same coalescence which the fat generally has in the body. It became very soft when exposed to the heat of a fire, and penetrated through paper on which it was spread, and rendered it more transparent. This paper burned with considerable crackling.

"The hair with which the fatty substance was mixed, grew out of the inner surface of the capsule containing it; in some places in solitary hairs, but chiefly in small fasciculi, at scattered, irregular distances. Besides these, there were loose hairs involved in the fatty mass. Some of the hairs were of considerable length, even to three inches; they were fine, and of a light-brown colour. They much more resembled the hairs of the head than those commonly on the pubes, and they corresponded very much in colour to the hair of the girl's head.

"There arose also from the inner surface of the capsule some vestiges of human teeth. One of these appeared to be a canine tooth, one to be a small grinder, and two to be incisors; besides, there was also a very imperfect attempt at the formation of another tooth. These teeth were not fully formed, the fangs being wanting; but in two of them the bodies were complete. Each of them was enclosed in a proper capsule, which arose from the inner surface of the ovarium, and consisted of a white, thick, opaque membrane.

"There was a white, spongy substance attached to the capsules of three of the teeth. The membrane of the ovarium itself was of considerable thickness, but unequal in different parts; its inner surface was very smooth, and its external surface irregular. The uterus was smaller than it is commonly at birth; it was perfectly healthy in structure;

and on opening its cavity it exhibited the ordinary appearances of a uterus at the age of the child. The left ovarium was very small, corresponding to the state of the uterus. It clearly appears from this circumstance that the uterus had not yet received the increase of bulk which is usual at the age of puberty. The hymen was entire, and such as is commonly found in a child of the same age; a lanugo was just beginning upon the labia, not more than what is often found on the upper lip of a boy fifteen years old. Such are the circumstances of this singular case, and they present to the mind various points of consideration.

"The formation of hair and teeth is a species of generation; for, in fact, it makes a part of it, and strikes the mind as being very different from any irregular substance which is formed by a disease. This formation, too, takes place in a part of the body which is subservient to generation, and where a complete fœtus is sometimes formed. These facts corroborate the idea that the production of hair and teeth in the ovarium is a sort of imperfect impregnation. But when we take another view of the subject, there are reasons at least equally strong for believing that such productions may arise from an action in the ovarium itself, without any stimulus from the application of the male semen."[1]

Dr. Baillie stated, in the 'Morbid Anatomy of the Human Body,' published in 1797, that he had met with the same kind of fatty substance intermixed with hair, and the body of one tooth covered with enamel, in the ovarium of a woman about eighteen years of age. "In this case the uterus was rather less than its usual size in the adult when unimpregnated, and there was no membrana decidua formed in its cavity. It appeared, therefore, to be undergoing no change similar to what happens when there is an ovum growing in the ovarium or the Fallopian tube. The hymen, too, was perfect; the edge of the membrane being quite sound and natural, and the aperture in it being remarkably small. These circumstances do not amount to demonstrative evidence, but still must be considered as a very strong confirmation of the truth of the opinion above stated."

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[1] 'Philosophical Transactions.'

'The Works of Mathew Baillie, M.D.,' by James Wardrop, vol. i, p. 139. London, 1825.

The first volume of the 'Medico-Chirurgical Transactions' contains a paper by Mr. Abernethy, entitled, "An Account of an Uncommon Disease of the Ovarium." This was an example of an ovarian cyst, similar to those which Dr. Baillie had described; and Mr. Abernethy observes that "this case is curious rather from the rarity of its occurrence than from its nature. A similar fluid is secreted by the sheaths of the tendons and the bursæ mucosæ."

"I have in my possession," observes Dr. Merriman, "a preparation consisting of the ovarium enlarged, and filled with fatty matter, intermixed with hair, which has formed to itself a bed between the rectum and vagina. Had the woman from whom these parts were taken become impregnated, the ovarium must of necessity have been confined in this situation, and a tumour occupying a considerable space must, in consequence, have been formed in the pelvis, which would have proved a great obstacle to parturition."[1]

Two fatal cases are referred to by Dr. Merriman, in which difficult labour arose from the presence of ovarian cysts in the pelvis filled with fatty matter, intermixed with hair and teeth.

The ninth volume of the 'Medico-Chirurgical Transactions' contains "An account of a case in which some singular preternatural appearances were observed in the Ovarium and Female Bladder," by Dr. Edward Phillips. The patient was thirty years of age, and "from early life she had, at different periods, found a difficulty in passing her urine;" "but these attacks were but slight and transient." About two years before her death she was seized with inflammation of the bladder, and a fulness appeared in the left side of the hypogastric region, and afterwards "a large, indurated tumour, somewhat oblong in form, extending from the situation of the spleen to the umbilicus."

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[1] 'Med.-Chir. Trans.,' vol. iii, p. 52, 1816.

"On opening the cavity of the abdomen, there escaped about two gallons of water mixed with blood. On the left side of the umbilical region, there was an ovarial tumour, rather larger than a human heart; the contents of which were a semifluid substance, a good deal resembling in appearance clouted cream. In the middle of this cream-like substance was found a tuft of hair about the size of a hen's egg. The surface of the tumour was nearly covered by clusters of hydatids, beautifully transparent and concocted like a bunch of grapes. On the broad ligament of the left side of the uterus, there arose a number of small, white tumours of the size of common peas. The uterus itself was not diseased. The bladder was very much distended, or rather plugged up with a substance similar to that which was contained in the ovarial tumour; and here also was discovered another large tuft of hair. The coats of the bladder were very much indurated, and particularly the inner coat. The urethra appeared to have no direct communication with the bladder; at the under and posterior part of which there was attached a small cyst, having the same cream-like substance before described, and also a quantity of hair, and, what is deserving of particular notice, there was a perfectly formed incisor tooth, having the enamel, and its fang firmly attached to the coats of the cyst. This cyst or cavity had a communication with the bladder by means of three small foramina, and it communicated also with the urethra on the anterior part."[1]

In the tenth volume of the 'Medico-Chirurgical Transactions,' Dr. Merriman has related two more cases in which parturition was rendered difficult by the presence of these ovarian cysts between the vagina and rectum; and Dr. Bostock analysed the sebaceous matter taken from one of these, and found it to consist of "a concrete animal oil, nearly in a state of purity, similar to butter or lard."

It does not appear, from the 'Medico-Chirurgical Transactions,' that any communication has been made to this Society on ovarian cysts which contain teeth, hair, and fatty matter, during the last forty years. They have not, however, during this long interval, ceased to attract the attention of the most eminent pathologists; and when opportunities presented, they have been subjected to more or less accurate anatomical investigation.

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[1] 'Med.-Chir. Trans.,' vol. ix, p. 427, 1818.

"There are a great number of cases of this peculiar condition of the parts on record," observes Dr. Seymour; "and one well known, described by Dr. Baillie in the 'Philosophical Transactions.' A case also occurred recently in St. George's Hospital; a woman, about thirty years of age, some weeks after delivery, having been admitted under the late Dr. Young, with symptoms of enteritis, which speedily proved fatal. The inferior portions of the small intestines were found inflamed, which inflammation appeared to have been excited by the presence of a tumour of the size of a large cricket-ball, which had been attached by a narrow neck to the posterior part of the left ovarium. Its proper coat was of a fibrous texture, and of a purple colour, and enclosed a mass of sebaceous matter, penetrated throughout with long, fine hair; after removing which, a full-grown incisor tooth was found attached to the fibrous coat."[1] The preparation of the parts is now placed upon the table of the Society.

Dr. Seymour was strongly inclined to believe that these growths in the ovaria were the "result of an imperfect conception in the mother of the individuals in whom they are found."

M. Cruveilhier has published delineations, in his eighteenth fasciculus, of various cysts of the ovaria which contained teeth, hair, and fatty matter; and the following are the conclusions which he has drawn from his elaborate researches:

"Eh, bien! les kystes pileux ovariques sont également le resultat de l'adhésion de deux individus, savoir, du produit de la conception qui est resté féconde dans l'ovaire, et de l'ovaire lui-même. Cette adhésion se fait toujours avec fusion plus ou moins complète; si bien que des dents, des débris de mâchoires, des os plus ou moins informes, des lambeaux de tissu cutané et du cuir chevelu en particulier, garni de poils plus ou moins long, sont presque toujours le seul vestige de la presence antérieure d'un fœtus; et les kystes les plus compliqués sont l'explication toute naturelle des cas les plus simples, des cas de kyste ovarique, dans lesquelles on ne trouve que de la matière grasse et des poils."[1]

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[1] 'Illustrations of some of the Principal Diseases of the Ovaria,' &c., by Edward J. Seymour, M.D., 1830, p. 82.

Four cases have come under my observation, in which ovarian cysts were found containing hair and fatty matter. Two of these cysts were in a natural state, and afforded the best opportunities that could be enjoyed, to examine their structure, and mode of formation in the substance of the ovaria. The other two cysts were in a diseased condition, being affected with dropsy and cancer, or having their coats disorganized by inflammation, suppuration, and ulceration.

In this communication I propose—1st, to describe in a succinct manner the appearances presented by the two cysts which were in a natural condition; and 2dly, the two ovarian cysts which were in a pathological state.

I. On the 5th of February, 1853, Dr. Martin, of Arlington Street, presented to me for examination the uterus and its appendages, removed from a woman, aged forty-three—the mother of one child, aged twenty—who had died the preceding day from cancer of the uterus. The os and cervix were partially destroyed by ulceration, and there was a hard, fibrous tumour imbedded in the muscular coat of the body and fundus. The left ovarium was enlarged to the size of a child's head; and the Fallopian tube, much elongated and adherent to the ovarium, was filled with pus. Two cysts nearly equal in size were found in the left ovarium, one of which contained purulent fluid, and the other a fatty-looking matter, with a great number of hairs loosely imbedded in it, varying in length from two to six inches.

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[1] 'Anatomie pathologique,' par J. Cruveilhier, xxxvi livraison, p. 3, 1842.

The fatty matter, which was of a yellowish-white colour and of the consistence of honey, only adhered to the inner surface of the cyst at one point, and around this spot, and nowhere else, were hairs observed growing from the inner surface of the cyst. Some of these hairs were attached to the cyst by a root; but others through a considerable part of their length seemed to be attached at the middle part, both extremities being free and pointed. Some of the hairs found loosely imbedded in the fatty matter had two sharp-pointed extremities and no roots; others had one slender point, and the other extremity presented the appearance of having been cut across, without any bulb by which it could have grown from the cyst.

Some of these hairs have been preserved, and are now exhibited.

Dr. Bence Jones examined the fatty matter which filled the cyst; and stated that at the temperature of the living body, it must have been an oleaginous fluid nearly pure, which had assumed the degree of consistence which it possessed subsequent to the death of the patient and the cooling of the body.

The walls of the cyst were composed of three distinct coats. 1st, the peritoneal coat and a thin layer of the stroma or parenchymatous structure of the ovary; 2dly. a dense fibrous coat; and 3dly, a lining membrane, uniformly smooth, white, and glistening, except where the hairs and fatty matter were adherent to it, which portion was of a peculiar yellow colour, resembling the corpus luteum, and thick and hard.

II. On the 6th of October, 1833, I examined the body of a woman, who had died the previous day, the eleventh after delivery. Occupying the brim of the pelvis, there was an ovarian cyst of an oblong shape, and an inconsiderable size. An opening was made into the cavity of this cyst, which was seen filled with fatty matter, intermixed with hairs. To prevent the escape of the hairs and fatty matter, the opening in the walls of the cyst was closed; and the parts were suspended in alcohol, and placed in the museum of St. George's Hospital, where they remained without being examined until November, 1859, a period of twenty-six years. The preceding case, and two others about to be related, which had occurred subsequent to 1833, made me determine to subject the walls of this cyst and its contents to the most careful anatomical examination. Having placed the parts in a shallow vessel filled with alcohol, I proceeded, with my small forceps and curved needle, and the dissecting lens which magnifies six diameters, to investigate the structure of the cyst and its relation to the stroma and peritoneal coat of the ovarium. It appeared that the cyst was imbedded everywhere in the stroma or parenchymatous structure of the ovary, like a Graafian vesicle. Between the outer surface of the cyst and peritoneal coat of the ovary, there was everywhere interposed a stratum of the parenchymatous substance or stroma. At the uterine extremity of the ovary the stroma formed a thick stratum. On examining the cyst itself, I found that, like a Graafian vesicle, it consisted of two distinct membranes or layers; the outer thick and dense, and the inner thin and transparent. These two layers of which the cyst was composed, were separated from one another very readily by means of the forceps and needle. Everywhere the cyst consisted of two coats, surrounded by the stroma of the ovarium and the peritoneal coat. There was seen adhering to the inner membrane of the cyst, at the outer extremity of the ovarium, a body with a slender neck, about the size of a nutmeg, of a yellow colour, like the corpus luteum; it was hard, but not bony, when cut into. The surface of this yellow body presented the appearance of a portion of human scalp, from which there were seen growing a number of hairs. Some of these when torn out were found to have roots, and to be in all respects organized like human hairs.

There were no hairs seen growing from any other part of the cyst.

The preparation of the parts and an accurate drawing by Mr. Roberts are placed upon the table of the Society.

This cyst, like the ordinary Graafian vesicle, consists of two layers; it is imbedded in the stroma of the ovarium like a Graafian vesicle, and the yellow body is seen growing from the lining membrane of the cyst, and to be in the situation usually occupied by the unimpregnated ovum. It appears to be a conclusion demonstrated by the appearances that the cyst is an enlarged Graafian vesicle, and that the formation of hair and fatty matter has taken place within the cavity of this vesicle.[1]

By what process, teeth, bone, hair, and fatty matter are formed within the Graafian vesicle without impregnation, I will not attempt to explain, or to remove this hidden process from "*the domain* of the wonderful." In the works of the Almighty there are no freaks to be detected, similar to those often observed in the actions of men.

A collection of the hairs is placed before the Society, and the following is the account by Dr. Noad of the chemical composition of the fatty matter found in the cyst:

"The following are the results of my examination of the substance I received from you, through Mr. Hooper.

"As it came into my hands, it was a clammy, cheesy-looking substance, matted together with hair; it weighed 77·3 grains.

"When placed in the water-oven it soon melted; and after remaining for several hours at the temperature of 212° Fahr., the weight fell to 16·5 grains. When removed from the water-oven, it appeared to be a bunch of hair saturated with fat.

"It was washed into a beaker, and digested with repeated affusions of benzole; by this means the hair was obtained perfectly clean, and weighed, when dry, 2·5 grains.

"The benzole solution, after standing some hours, deposited a small quantity of a cheesy-looking substance, and the clear liquid yielded on evaporation 12·5 grains of a yellow fat.

"The matter insoluble in benzole weighed, when dry, 1·5 grain. It was divided into two parts; one part gave, by fusion with caustic potash, and subsequent treatment with hydrochloric acid, abundant evidence of the presence of *sulphur*; the other portion afforded all the reactions of an albuminous or protein compound.

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[1] See the Plate, in which the appearances here described have been represented.

"I regret that the quantity at my disposal was so exceedingly small, that I was unable to submit it to any further experiments.

"Roughly, however, the composition of the substance (as it came to me) may be expressed thus, in one hundred parts:

Water	78·60
Hair	3·23
Fat	16·17
An albuminous substance, having many of the characters of *casein*, and containing much sulphur	2·00
	100·00"

III. In November, 1842, I was requested by the late Mr. Gaskell to see Miss F——, aged twelve years, who had an enlargement of the hypogastrium. The catamenia had not appeared, and there were none of the symptoms of puberty present. About the end of October, 1843, the enlargement of the abdomen had greatly increased, and fluctuation was very distinct. On the 2d of November she was tapped by Mr. Aston Key, and a quantity of dark-coloured, gelatinous fluid, evidently the product of an ovarian cyst, was drawn off. After the fluid had been drawn off, the lower part of the abdomen was still hard and irregular, and a solid mass, the size of a hen's egg, was distinctly felt, the day after the tapping, in the epigastrium.

In July, 1845, the fluid had again collected, and the abdomen was very large. It was then proposed, by several medical practitioners, that the operation of ovariotomy should be performed; but, at my suggestion, recourse was again had to tapping. During the tapping, the canula being obstructed, the fluid ceased to flow, and on inquiring into the cause of this, it was discovered to have arisen from a quantity of fatty matter and long hair. It was at once obvious that the dark-coloured, viscid fluid was flowing from a cyst which contained hair and fatty matter, and probably a jaw-bone and teeth.

The patient subsequently died at Ramsgate; and it was stated to me, that there was found in the abdomen a great mass of multilocular cysts and cancerous structure, with which were interspersed numerous long hairs and pieces of bone. The portion which I had an opportunity of examining left no doubt upon my mind that the stroma or parenchymatous substance of the ovarium had become affected with cancer, and that the cyst which contained the hair, bone, and fatty matter had become dropsical.

Dr. Cleghorn relates the case of a woman, who died ten days after being tapped. The right ovarium was found greatly enlarged, and had many cells containing hair, cretaceous matter, fragments of bone; and teeth; others gelatinous fluid.[1] This case has been referred to by Dr. Burns, as well as another mentioned in the 11th vol. of the 'Physical and Literary Essays,' at p. 300; in which the one ovarium contained many vesicles—the other contained a mass like brain, with bones and teeth.

IV. On the 9th April, 1858, Mrs. C—, æt. 28, in the fifth month of her first pregnancy, arrived in London from Singapore; and on the 10th consulted Dr. Allan, of Hyde Park Place West. She was in an exhausted condition, passing large quantities of pus with the urine. Dr. Allan received the following history of the case, drawn up by Dr. Little, of Singapore, her last medical attendant:

"I was called to see Mrs. C— about the end of 1855, and found that, although she was of spare habit, yet she had an abdominal protuberance which made her appear as if she were seven months pregnant. On examination I found a tumour occupying the lower region of the abdomen, and rather to the left of the mesial line. It was hard, round, and slightly moveable, giving the idea of a tumour fully distended with fluid. The general health was not good, appetite spare, and none for breakfast. Bowels rather costive. Much weakness and lethargy were experienced, with a feeling of bearing-down from the presence of the tumour. She was occasionally attacked with intermittent fever and neuralgia of the face, having had, in India, severe attacks of ague. Mixed with the urine, but afterwards deposited, was a copious white matter, which, after careful analysis, I found to be purulent. The history of the case was that Mrs. C— was married on the 25th of July, 1853. For some time previous she had been irregular in menstruation, both as regards time and quantity, and always it had been attended with pain. One month after marriage she noticed a tumour in the abdomen, which rapidly increased in size, and was attended with much pain and tenderness on pressure. On reaching Calcutta one medical gentleman pronounced her five months *enceinte*; another that she had a fluid ovarian tumour. She then visited Penang in September, and in October or November noticed certain movements in the tumour which were painful and startling. Lady friends there thought her in the family way; but the medical practitioner who attended pronounced the tumour ovarian. Long after (but the exact time not known) the movements had ceased, she observed a white discharge in the urine, which continued until I saw her in November, 1855. Seven months after marriage the menstrual discharge had returned, and has continued pretty regular as to time until within the last four months, during which it has not appeared. After a careful consideration of the case in 1855, I came to the conclusion that Mrs. C— had an ovarian tumour of a fluid nature, which was discharging itself by the bladder. At first I thought the pus was excreted from the kidneys; afterwards that there existed a communication betwixt the sac and the bladder. My prognosis was that the sac was gradually emptying itself in a most unusual way, and that, if the evacuation could only exceed the secretion, the patient would recover. My treatment consisted in strengthening the general system by tonics of iron and quinine; subduing local irritation by fomentations, leeches, and blisters, especially over the left ovary, where pain and tenderness were often complained of; encouraging the flow of the menstrual fluid; and maintaining gentle pressure over the tumour and abdomen generally by an elastic bandage. Occasionally I had to prescribe a mild alterative for biliary derangement, and frequently laxatives. Under this treatment Mrs. C——'s health improved very much, and the tumour diminished slowly, but perceptibly, until July, 1857, when severe pain was felt in the neck of the bladder and meatus urinarius, with incessant calls to micturition. After this, profuse discharge took place from the urethra, to the amount of four tumblers of pus daily. This gradually diminished to seventeen tumblers in the month of October; eleven and a half in November; six in December, 1857; and now, in January, 1858, there is little more than one ounce daily. The discharge varies in consistence—thin at one time, at another thick and greenish, and of a most offensive odour; but the first was of thinner consistence than the last.

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[1] 'Transactions of the Royal Irish Academy,' vol. i, p. 80.

"To the astonishment of the lady, and to my surprise, one day, a large mass of hair, evidently fœtal, was abstracted from the urethra, affording great relief; and at future times several such masses of hair were abstracted, as well as small shreds of bone. I now, for several reasons, arrived at the conclusion that the communication with the sac was not into the bladder, but into the urethra; and that the tumour was not an ovarian cyst, but an extra-uterine conception, which was gradually discharging itself by the urethra, having been by utero-decomposition completely broken up. The feelings of the patient, and the abdominal movement while in Penang, were no doubt fœtal; and had the stethoscope been judiciously employed, the fœtal sounds must have been heard, and all doubt as to the case removed. But the opportunity was allowed to pass by, and the fœtus died in the sac in the abdominal parietes; and when I first examined the tumour, every symptom indicated an ovarian cyst, and it was not until the hair and the small fragments of bone presented themselves, that I acknowledged the movements felt in Penang to be fœtal, and the case to be one of extra-uterine conception.

"At present, in January, 1858, the tumour is felt in the lower region of the abdominal parietes about the size of a large lemon. It shifts sometimes to the right or left of the mesial line, and increases and diminishes according to the collection of matter inside, which now averages ten ounces daily. Some urethral irritation is still felt, but nothing to what has been. With the matter a large proportion of clotted blood passes, and occasionally a hair or two. For several months no menstrual discharge has passed, which, with the enlarged breasts, darkening of the areolæ, and a fulness about the uterine region, makes me suspect that the patient may be again in the family way.

"The treatment during the last six months has been to allay local irritation, subdue general excitement, and counteract the depressing effects of the excessive and putrefactive discharge. In addition to local fomentations, the patient was taught to introduce the catheter into the sac to draw off the discharge, and clear out the sac with warm water and tincture of opium—latterly with warm water and a stimulating astringent injection composed of matico, myrrh, and lavender, with sulphate of zinc. I intended, when the hair had ceased to come away, and symptoms had shown that the contents had been discharged, to have injected with tincture of iodine, so as to have closed the sac; but owing to Mrs. C—'s departure, that, or any other means, must be done by others. Before any decided reduction of size had taken place, I recommended Mrs. C— to make three measurements of the abdomen. The highest, round the waist, and meeting at the umbilicus, was thirty-one inches and a half. The same was, in January, 1858, reduced to twenty-seven and a quarter. The second, passing above the hips, and meeting between the umbilicus and pubes, was reduced from thirty-four inches and a quarter, to thirty-two inches and a half. The third, meeting over the pubes, fell from thirty-six inches and a quarter to thirty-three inches and a quarter. In short, Mrs. C—, while six months ago she had the appearance of being seven or eight months pregnant, has now no appearance of any abdominal enlargement."

"Dated Singapore;
25th January, 1858."

From the 10th of April, 1858, to the 21st of May, Mrs. C— was under the care of Dr. Allan, and her sufferings during this period were intense. The pain she experienced chiefly arose from the passage through the urethra of numerous hairs coated with calcareous matter, a collection of which calculi, made by Dr. Allan, accompanies this communication. Mrs. C— stated to Dr. Allan, that while at Singapore, similar sharp-pointed and curved calcareous concretions were passed from the bladder, which led Dr. Little to conclude that there had been an extra-uterine conception, and that these were the bones of the fœtus which had been "by utero-decomposition completely broken up."

At one o'clock in the morning of the 21st of May, 1858, Mrs. C— was seized with puerperal convulsions. At two p.m. I saw her, in consultation with Dr. Allan; and at three, the fits continuing with great violence, we agreed that delivery was the only means that could be employed to save her life. From the extreme rigidity of the os uteri, the delivery was not accomplished without the greatest difficulty; but we at last succeeded by our combined efforts in emptying the uterus. No convulsions followed the delivery, and consciousness returned; but she died two days after.

I obtained permission, with the utmost difficulty, to examine the body; and did so in the presence of Dr. Allan and Dr. Bence Jones. The preparation of the uterus, ovarian cyst, and bladder are now placed on the table of the Society. It will be seen that the uterus is in a healthy condition—that there is an ovarian cyst with thick walls on the left side, and that hairs are seen growing at one point from the lining membrane of this cyst—and that there is a considerable opening, into which a bougie has been passed, between the cyst and the urinary bladder. The right ovarium displaced contained the corpus luteum. None of the hairs in the cyst were coated with calcareous matter; but all those found in the bladder, and all that had escaped through the urethra during life, were encrusted with ammoniaco-magnesian phosphates. This is stated on the authority of Dr. Noad.

APPENDIX TO THE ABOVE PAPER.

Received March 10th.—Read March 13th, 1860.

In the autumn of 1859, I related the history of the last of these cases to John North, Esq., late Lecturer on Midwifery at the Middlesex Hospital. Mr. North then informed me, that about twenty years before, he had under his care a young lady who had calculus of the bladder, which was extracted by Sir B. Brodie, and that with the fragments of the broken calculus there were a small portion of bone, of an irregular shape, and two imperfectly formed human teeth. Mr. Charles Hawkins subsequently brought to me the 'Lectures on Diseases of the Urinary Organs,' by Sir B. Brodie, published in 1849, where I found, at p. 262, the following account of this and other cases:

"In the year 1840, I was consulted with Mr. North respecting a young lady labouring under very aggravated symptoms of calculus of the bladder. The existence of the calculus having been ascertained, I proceeded to extract it by an operation; the steps of which I shall describe hereafter. On examining the bladder with the finger, previously to the introduction of the forceps, I ascertained that the calculus was adherent to it at the fundus. However, I seized it with the forceps, and with the application of a moderate degree of force, was enabled to extract it, though not without it having been broken into several pieces. These fragments were found to consist chiefly of the mixed phosphates; but among them were a small portion of bone, of an irregular shape, and two imperfectly formed human teeth. If you refer to the tenth volume of the 'Medico-Chirurgical Transactions,' you will find a case and dissection recorded by Dr. Phillips, which fully explains the nature of the case in question. The teeth and bone, being the result of an original malformation of the ovarium, had become attached to the mucous membrane of the bladder, and formed the nucleus on which the calculous matter had been deposited. A similar case occurred in the practice of Mr. Warner, and is described in the forty-ninth volume of the 'Philosophical Transactions.'"

Mr. North has since called my attention to the following note at p. 456 of Dr. Hemming's translation of 'Boivin on Diseases of the Uterus,' published in 1834.

"In this manner a cyst of the same kind opened into the bladder, and for a long time allowed the hair to pass with the urine; at last a body was extracted from the bladder, as large as a hen's egg, presenting at one of its extremities a shred of skin, containing hairs and a bone; in which was partly fixed a kind of tooth, resembling a small molar. The communication of the cyst with the bladder was ascertained by the finger passed into the urethra. The person recovered. (Delpech, 'Chirurgie Clinique,' tome ii, p. 521.) In a similar case, Dr. Paul Manhall observed, on post-mortem examination, that the ovaria were united together into a cerebriform or fatty mass, containing an extraordinary quantity of hairs and fine teeth. The cavity which contained them communicated with the bladder." ('Journal complémentaire,' tome xxxv, p. 183.)

I related to the President of the College of Surgeons the last case detailed in my paper, and showed him the collection of calculi formed upon the hairs which had passed from the ovarian cyst into the bladder, and through the urethra during life. He had not met with any similar case. He described what he had seen, the same evening, to Mr. Lawrence, and this led to the following very interesting letter being communicated to me by Mr. Lawrence on the 9th of March, 1860, a considerable period after the paper had been presented to the Society.

"Whitehall Place;
"March 9th, 1860.

"My dear Lee,—Not having preserved any memoranda of the case I mentioned to you, in which calculous matter in a fragmentary and loose condition, intermixed with hair, had been removed from the female bladder, I can send you only such an imperfect account as can be supplied from memory, after the lapse of nearly half a century.

"The patient, a married lady and mother of three children, who had reached adult age some years previously, must have been nearer seventy than sixty years of age when she underwent the operation of lithotomy by Mr. Abernethy. One of her daughters, a single lady, who is now eighty-five, remembers the facts of her mother's case most minutely, and has kindly communicated them to her medical attendant, who has been so obliging as to favour me with the following narrative: 'Before the late Mrs. — was married even, she had some peculiar illness in Bath, and was under the care of Dr. Moisey, when an abscess formed connected with that part of the body. At some time previous to the attendance of Mr. Abernethy and yourself, she had peculiar symptoms. On one occasion Dr. Baillie attended her; she had pain in the side; Dr. Baillie said it was liver. Leeches were applied; she became easier, and a small stone passed away. At the time of Mr. Abernethy's attendance, as much as a small saucer-full came away; many of the stones were as large as horse-beans, and there was hair amongst them. She says that you remained twelve or fourteen nights, and that on one occasion her mother told you she could jump up and run away. She became perfectly well after the operation, without any degree of incontinence or difficulty in retaining the urine.

"'The operation was performed in 1816, and death occurred in 1819. At some intermediate time the calculus which accompanies this was passed. The patient was very uneasy in the night; her daughter got up and gave her the chamber bed-utensil called a slipper. As the water passed, something hard was heard to drop into the vessel, and proved to be the calculus sent herewith. In the latter part of life the urine was frequently very fetid, thick, and sometimes ropy, so that death was probably hastened by the irritation of disease in the urinary organs.'

"My recollection of what was removed in the operation is that it consisted entirely of separate calculous portions, the largest being of the size mentioned in the narrative, the remainder gradually lessening down to minute particles. The latter were mixed with short hairs, so as to constitute a softish mass. There were several separate incrusted hairs.

"I remain, yours very faithfully,

"WM. LAWRENCE.

"Dr. R. Lee."

POSTSCRIPT.

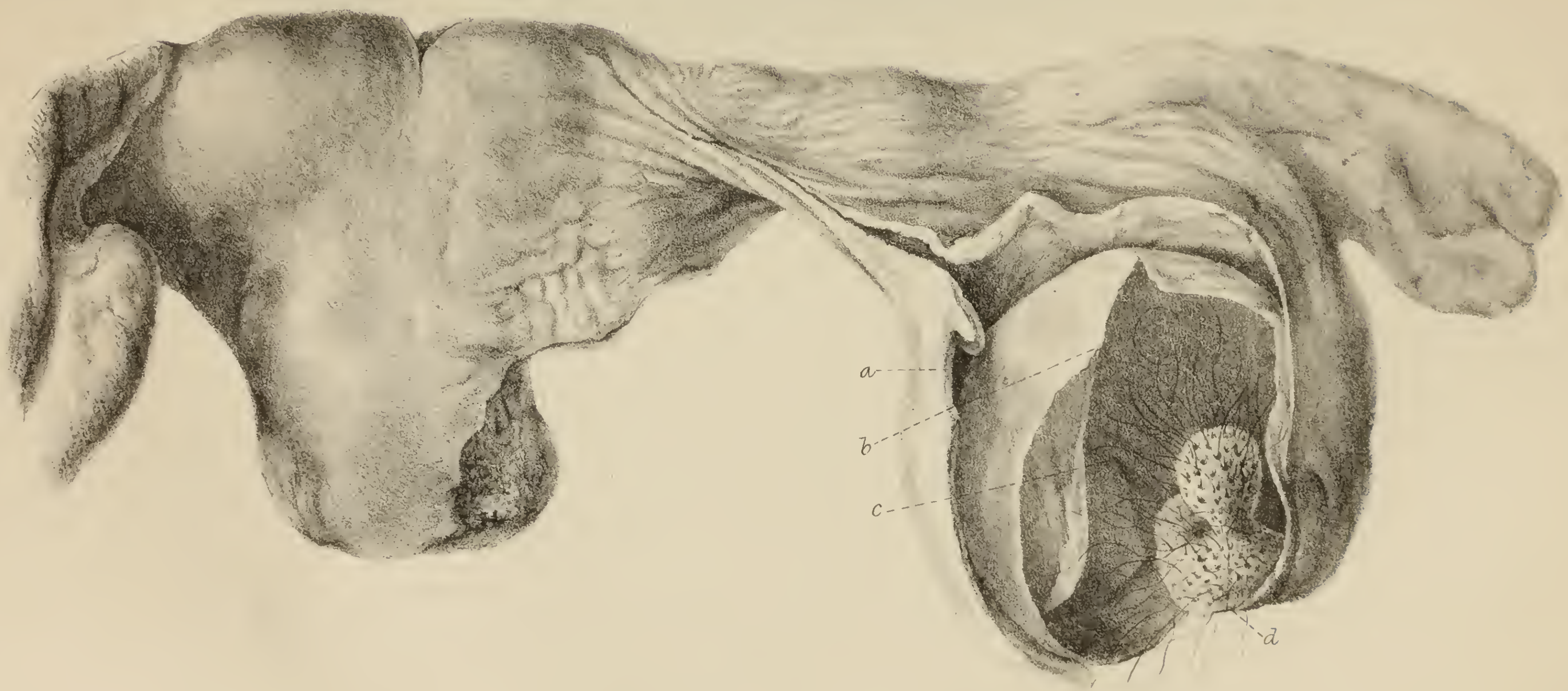
Reference ought to have been made at p. 97 of this paper to a case detailed in the thirty-fourth volume of the 'Medico-Chirurgical Transactions,' entitled "An Account of a Dissection of an Ovarian Cyst which contained Brain, by Henry Gray, F.R.S."

EXPLANATION OF THE PLATE.

The drawing is an illustration of the first case recorded in the paper, and represents the uterus with the right ovarium, which contained the cyst.

a. The peritoneal covering and stroma of the ovary.
b. The fibrous coat.
c. The lining membrane of the cyst.
d. Point of attachment of hairs and fatty matter.

Plate VI



G. H. Ford lith.

W. West imp.

CASES
OF
INTRA-UTERINE FRACTURE,
WITH OBSERVATIONS
TO SHOW THE ANALOGY BETWEEN FRACTURE IN UTERO AND CONGENITAL DISTORTION.

BY

BERNARD E. BRODHURST, F.R.C.S.,
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Received Jan. 2d.—Read March 27th, 1860.

THERE are several instances of fracture of the bones of the fœtus on record—instances both of simple and of compound fracture. Thus, Montgomery states: "I saw a woman, eight months pregnant, fall from a window twenty-five feet into the stony street, on her face; her hip-joint was dislocated, and her face and hands cut, but the uterus was not ruptured. She was delivered that night of a dead child, which had some of its bones broken, and had sustained several other injuries. She recovered perfectly."[1] There are also numerous instances of fracture of the fœtal skull on record, as well as of depression of the cranial bones.

Such cases are related by Montgomery,[2] West,[3] W. J. Schmitt,[4] Meissner,[5] Schnuhr,[6] D'Outrepont;[7] and by

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[1] 'An Exposition of the Signs and Symptoms of Pregnancy,' Case 42, p. 680, 2d edit., 1856.

[2] Ibid.

[3] 'Med.-Chir. Transactions,' vol. x, 2d series, 1845.

[4] 'Abhandlungen der Physikalisch-Medicinischen Societät zu Erlangen,' Band ii, s. 60, 1812.

[5] 'Kinderkrankheiten,' Band iii, 1826.

[6] 'Medicinische Zeitung von dem Verein für Heilkunde in Preussen,' N. 32, s. 152, 1834.

[7] 'Neue Zeitschrift für Geburtskunde,' Band ii, s. 116.

Osiander, Roederer, Schilling, Flügel, Baudelocque, and others. Rokitansky says, speaking in reference to "Anomalies and Diseases of the Osseous System:" "There is a remarkable form of incomplete fracture, in which a bone becomes bent. It occurs on flat bones, like the skull, as well as on long bones. It is produced sometimes by sudden and violent mechanical force, sometimes by more gentle means, which act through a longer period, either uniformly or with intermissions. Its occurrence is favoured by the softness of the bones, which in fœtal life and in childhood exists naturally, but in subsequent periods of life is a morbid condition. These inflections of bone are chiefly observed in the skull of the new-born child, as a consequence of the pressure which the head has undergone from the pelvis of the mother or the forceps of the accoucheur. They may, however, be occasioned by accidental or intentional violence after birth, or they may take place in the bones of the limbs of persons who are affected with rickets or osteomalacia. They may be brought on by mechanical violence or by excessive muscular contraction."[1]

There are also cases, such as those to which allusion has just been made in the last quotation, where the skeleton is cartilaginous or very imperfectly ossified at birth, and where solutions of continuity have been found. Such a case is related by Sartorius[2] as congenital rickets. The same case, however, has been quoted by Graetzer[3] and others as one of intra-uterine fracture. Other cases of the same kind are reported by Amand,[4] Chaussier,[5] Mansfeld,[6] Barker,[7] D'Outrepont,[8] and others, as instances of intra-uterine fracture. Thus, allusion has now been made to intra-uterine fractures where the bones are healthy, to fractures which have taken place during labour, and to solutions of continuity through congenital rickets. But the cases to which I desire to invite the attention of the Society this evening are those where, fracture in utero having occurred, reunion, more or less complete, has been found at birth; or where the appearance of the wound has been such as to lead to the conviction that fracture had occurred some days or weeks before birth. These cases are rare, and, inasmuch as they are scarcely alluded to by British authors, I may be allowed to cite some few cases before I record two instances of a similar character which have lately occupied much of my attention.

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[1] 'A Manual of Pathological Anatomy,' p. 141, vol. iii, Sydenham Society's edition.

[2] 'Rhachitidis Congenitæ Observationes.'

[3] 'Die Krankheiten des Fœtus,' 1837.

[4] 'Nouvelles Observations sur la pratique des Accouchemens,' obs. 8, p. 92, 1714.

[5] 'Bulletins de la Faculté de Médecine de Paris,' No. 3, p. 306, 1813.

[6] 'Journal der Chirurgie von Gräfe und Walther,' Band xix, s. 552, 1833.

[7] 'British Medical Journal,' Sept. 26, 1857.

[8] 'Abhandlungen und Beiträge Geburtshülflichen Inhalts,' s. 233, 1822

Professor Ploucquet, of Tubingen, writes thus: "A pregnant woman fell on the abdomen, and gave birth, seven weeks after the accident, to a seven-months child. It was observed, at birth, that the left femur was upraised and deformed in the middle of its shaft. The child died twelve days after birth, when it was seen that the broken ends of the bone had overlapped, and had thus united firmly, and that callus was thrown out around them."[1]

The following case is related by Kopp: "A woman, æt. 30, whilst in the thirty-fourth week of her first pregnancy, fell into a pit, where she remained lying and unable to move. For many weeks after, and indeed until the termination of pregnancy, she experienced pain on the left side of the abdomen. The movements of the fœtus were not felt after the fall. Six weeks after the accident she gave birth to a boy; it was a normal, head presentation, and the birth was easy and rapid: it took place without instrumental interference. The right leg was short and misshapen, the tibia having been fractured near the middle of the bone. At birth there was a wound which corresponded to the fracture, and from which a slight serous discharge exuded. On examination, seven months after birth, a callus was found around the tibia and the fibula."[1]

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[1] Loder's 'Journal,' Bd. ii, s. 782, 1800.

Devergie read the following case before the Académie Royale de Médecine, February 24th, 1824 :—"Fracture and reunion of the Clavicle in a Fœtus in Utero.—A woman, in the sixth month of pregnancy, fell from a high seat, and struck the abdomen forcibly against the angle of a table. The pain was excessive, and lasted for a considerable time without abatement. At length, however, it ceased, and at the ordinary period of parturition a healthy child was born, but with a considerable swelling on the left clavicle. The child died on the eighth day, when, on examination, a firmly reunited fracture of the clavicle was found, the ends of the bone slightly overlapping." The preparation, he goes on to say, is in the museum of the hospital of Val de Grâce.

Carus relates, that "a strong young person, a servant, named Caroline Henriette W—, æt. 25, on the 31st of March, 1826, fell on the abdomen, being then in the sixth month of pregnancy. Immediately after the accident violent movements of the fœtus were felt; from that time, however, they became fainter and fainter. At the usual time a small, ill-nourished child was born; it was feeble, and showed but indistinct signs of life. On the right leg was a transverse wound, three fourths of an inch in length, extending from the outer to the inner malleolus, and corresponding to it was a fracture of the tibia. The fractured extremity of the bone appeared in the wound, and was directed outwards; the periosteum was destroyed, and the lips of the wound were pale and flabby. The broken bone was replaced, but the edges of the wound fell into a state of sphacelus; necrosis of the tibia took place, and the child died on the thirteenth day."[2]

Schubert mentions that a child was born with compound fracture of the left femur. The bone protruded fully one inch, and it was carious. This child was one of twins.[1]

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[1] 'Jahrbücher f. d. Staatsarzneikunde.'

[2] 'Gemeinsame Deutsche Zeitschrift für Geburtskunde,' Band ii, s. 31, 1828.

Hofmedicus Sachse states that "a healthy peasant, æt. 42, who had already given birth easily to eight children, fell, whilst in the third month of pregnancy, on the ice, striking her left side, without, however, causing much pain. She had already felt the movements of the fœtus for a week. When she was rather more than five months advanced in pregnancy she fell from a ladder, and again struck her left side, against a block of wood. When she stood up after the accident, she felt a pricking pain in the lower part of the abdomen, and this pain became insupportable when she was recumbent and on her side. Blood escaped, *per vaginam*, on the third day, but abortion did not take place. For eight or ten days the movements of the fœtus were not felt, and when they returned they were accompanied with pricking pain, which pain lasted until the termination of pregnancy. At the full period of gestation a small and feeble, but living, child was born. There was an oblique compound fracture of the tibia and fibula of the right side, the lower ends of the broken bones being drawn upwards. The uterus had been wounded at each movement of the fœtus by the projecting bone; hence the pain which had been excited. At birth the fracture had reunited. The fractured leg was one inch and three fourths shorter than the other, and it was half an inch smaller in circumference. On the foot were only three toes. Also, the right arm, which had likewise suffered, was smaller than the left, and on the hand there were only three fingers."[2]

I am indebted for the following case to Dr. Richardson. It occurred in the practice of Dr. Moffat, of Hawarden, and the details were sent to Dr. Richardson, for his Fothergillian Prize Essay, 'On the Diseases of the Fœtus in Utero.'

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[1] 'Archives Générales de Médecine,' tome xvi, p. 288, 1828.

[2] 'Hufeland's Journal der praktischen Heilkunde,' Band lxiv, s. 27, 1827.

"On May 10th, 1846, I was requested," writes Dr. Moffat, "to visit Mrs. E—, who was in labour. On the night of the 4th of April previous, on going over the snow, she slipped, and fell with all her weight upon her right side, on the upper rail of a stile, over which she was climbing. The fall gave her so much pain that she could not move from the spot for a considerable time. The pains were, as I infer from her description, uterine. After she had rallied she walked home, and had to remain in bed for a few days. There was considerable ecchymosis on the right side of the abdomen. Ultimately she completely recovered, and went on well to the full period of utero-gestation.

"The labour was natural; it was a first pregnancy. When I was satisfied that the mother was doing well and was safe from immediate danger, the nurse directed my attention to the child, whose right arm was not, as she said, all right. I found a swelling on the lower third of the humerus, and on examination detected crepitus. There was here an ununited fracture, and the enlargement, I concluded, was produced by osseous matter. On examining the forearm, another swelling was detected, but union was complete. In the lower extremities I found the right much enlarged by osseous tissue, in the same manner as though it also had been fractured. I found also, on measurement, certain remarkable differences. The length of the sound arm, from the acromion of the scapula to the external condyle of the humerus, was three inches. This space in the fractured arm was two inches and a half. The girth of the sound arm was two inches and three fourths; that of the fractured arm at the fractured part was five inches. The length of the sound forearm was two inches and one fourth; of the fractured forearm two inches. The length of the healthy thigh, from the trochanter major to the external condyle, was three inches and a half; its girth was three inches and three fourths. The length of the injured thigh was three inches and one fourth; its girth at the fractured part was five inches and three fourths."

Case 1.—In February, 1858, Mr. W. H. Gardner asked me to see, with him, a young lady, æt. 13, of whom the following is the history. About three weeks before giving birth to this child, her mother fell down a flight of stairs: she struck the abdomen in falling, and was much hurt. She was at that time in the seventh month of pregnancy; uterine pains were felt, and it was thought that parturition would take place: the child was not born, however, until three weeks after the accident. At birth it was observed that there was considerable swelling about the right knee, which increased somewhat, and in the course of a fortnight discharged a small quantity of purulent matter. There was also a second swelling, in the neighbourhood of the trochanter, which, however, subsided without opening, and the bone in this position did not appear to be affected. At the knee, however, it was remarked, as the swelling subsided, that an abnormal condition of the bone existed. It was soon found that the leg could be only partially flexed, and that it could not be fully extended. Both the thigh and the leg were somewhat smaller than on the opposite side. When the child began to walk, the knee was thrown inwards, and the foot was directed outwards. The leg was soon found to be shorter than the other; and in consequence, the child wore a thick sole to her boot, to compensate for the deficiency in the length of the leg. Distortion, notwithstanding, increased. It was therefore recommended that artificial support should be given to the leg; and a steel support, from the hip to the foot, was consequently worn from her fourth year until I saw her.

On examination, I found that the foot was six and a half inches from the ground, that the knee was much inverted, and that the foot was directed outwards. The limb was much smaller, as well as shorter, than the other, and the motion at the knee-joint was very limited; the leg could not be fully extended, and it could not be flexed to a right angle. Further, there was no power to raise the foot, in extension, higher than three inches, and even at that height it could only be maintained for a minute or two. The thigh, however, could be well flexed on the pelvis. Without support, a single step could not be made. The muscles of the calf of the leg were contracted, so that the foot could not be flexed to a right angle.

It appeared to me that there had been a fracture through the epiphysis, separating the inner condyle of the femur, which, being somewhat rotated outwards, projected in front of the shaft of the femur. In consequence of the insufficient support for the head of the tibia, the internal lateral ligament had yielded, the knee had become inverted, and the hamstring muscles had become contracted.

Mr. Fergusson saw this case in consultation, and expressed his belief that it was probably an instance of intra-uterine fracture. It was determined to divide the tendons of the hamstring muscles and the tendo Achillis: which having been done, the leg was gradually extended, and the foot was flexed; the inversion of the knee also was overcome, so that considerable length was added to the limb; and, with some addition to the sole of the shoe, this young lady is now (nearly two years having expired) able to walk without artificial support and without limping. Before the tendons were divided she never attempted to walk, even in the house, without a stick; but now she never uses a stick, nor does she use artificial support except when she walks farther than the garden of the square in which she resides.

Case 2.—In December, 1858, I saw, with Dr. Gream, some few hours after its birth, a puny child, which was so starved in appearance that its chances of life seemed to be very small. The trunk and head were well formed, but the forearms and legs, and the hands and feet were abnormal. The ulna in each arm was wanting in its lower half, and the fibula of each side was also wanting in its lower half. The radius of either side was much bent inwards, and the tibiæ had been fractured at the junction of the middle with the lower third of each bone. These bones had been broken obliquely, and the skin had been wounded at the seat of fracture. At birth, the fractures had firmly reunited, and the skin was slightly adherent at the seat of fracture. Reunion had taken place irregularly, the lower portion of the fractured bone having been drawn upwards. A sharp projection was thus formed, which seemed to endanger the covering skin : this was especially the case on the left side. There was double talipes varus, and on each foot were only four toes; the fifth, or little, toe being absent. On the right hand were three fingers, *i. e.* a thumb, index and middle fingers ; the nails of the two fingers were fused, so as to form a single nail with a central depression, which corresponded to the web of the fingers. The fingers and thumb were webbed to their very tips. On the left hand there were only two fingers, the index and middle fingers, which, also, were webbed, as in the right hand. In each hand, carpal and metacarpal bones corresponded to the fingers. The left hand was drawn outwards by the action of the muscles. The radius and the metacarpal bones were shorter on the left than on the right side.

With regard to the probable causes of the deformities in this child, it may be mentioned that, during the fourth month of pregnancy, the mother fell, without, however, materially injuring herself, in running down a steep descent in Switzerland; and, again, that in travelling to London from the seacoast, the engine broke down and was detained in a railway tunnel for one hour, and, whilst in this position, the express train ran into that in which this lady was seated. A severe concussion was the result, but, beyond the shake and the fright, she was not injured. This accident occurred six weeks before parturition. The child was born at the full period of utero-gestation.

I may mention that, after the tibial tendons had been divided for the removal of varus, I endeavoured, by means of moderate pressure and the use of splints, to straighten the leg-bones. I found, however, that very little impression was made on them, but that the tendo Achillis was rendered extremely tense. It was only after this tendon had been divided that the bones could be efficiently acted on.

It must be admitted, I think, that intra-uterine fracture may occur as an effect of physical injury. Evidence appears to be strongly in favour of this point. Some have thought that these injuries to the fœtus are induced by *contre-coup.* But when we remember that the fœtus is surrounded by the liquor amnii, and that it receives a certain amount of support from the walls of the uterus, we shall probably incline to the opinion that the effect of coutre-coup on the fœtus in utero is impossible.

Distortion not unfrequently exists together with fracture. And, entertaining a confirmed opinion with regard to the origin of congenital distortions, Dr. Little[1] has expressed his belief that the origin of intra-uterine fracture and congenital distortion is similar. It has been contended by some that these fractures and distortions are produced by the pressure of the walls of the uterus. Thus, we find Scheffel,[2] in the middle of the last century, supporting this view. And, at an earlier period, Ambroise Paré[3] asserted an equally well-founded belief, namely, that congenital distortions were produced by the mother sitting cross-legged. Rudolphi,[4] however, showed that congenital distortions could not owe their origin to pressure in utero, since they were not unfrequently found to exist in the embryo at the third and fourth months. At this day his opinion prevails throughout Europe. If such, then, is the fact, that the limbs of the fœtus cannot be compressed by the walls of the uterus, we may discard also this theory, and assert, as of *contre-coup* so of compression, that fracture from one cause or the other is impossible, *so long as the membranes remain unruptured.* But if these causes are removed, we are thrown back upon the muscles as the only remaining cause of fracture and distortion. And here I would cite two instances of distortion, as illustrations of the mode in which abnormal muscular action may be excited in the fœtus. It will be seen that in these cases (which have long been published)[1] a somewhat similar cause gave rise to distortion as, in the cases already related, produced fracture. And this being the case, there can be no difficulty in admitting, with Dr. Little, that abnormal muscular action, however excited, is the cause, not only of congenital distortions, but of fractures in utero.

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[1] 'Lectures on the Deformities of the Human Frame,' p. 329, 1853.

[2] 'De Fœtu natibus in partu prodeunte, Observationes et Analecta,' 1770.

[3] 'Les Œuvres,' 1641.

[4] 'Grundriss der Physiologie,' 1823.

Case 3.—Early in 1852 the wife of a Lincolnshire farmer, returning home in the dusk of the evening, came unawares on the edge of a deep dyke, in which were from eight to nine feet of water, and into which another forward step would have carried her. She was alone at the time, and only saved herself from imminent destruction by a great and sudden muscular effort. Naturally, great emotion was excited by the danger and her escape from it. She was at this time in the sixth month of pregnancy. Until this occurrence she had felt the ordinary movements of the fœtus; from this time, however, they ceased, and she expressed her belief that her child was dead. On the fourth day after this event, however, a tremulous motion or slight fluttering was experienced, and this motion increased, until the movements of the fœtus were again felt, as before the accident. Parturition took place at the eighth month of utero-gestation: the child was very small and feeble, and it was affected with double club-foot and double club-hand. No other abnormality existed.

Case 4.—A robust woman, who was six months advanced in pregnancy, fell into an opening which had been left during the evening uncovered. She was hurt by the fall, but, after a short time, was able to proceed home. At the eighth month of utero-gestation she gave birth to a child, who had double club-foot and double club-hand. No other distortion or abnormality existed. This person had previously given birth to two healthy children.

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[1] 'On Club-foot and Analogous Distortions.' By B. E. Brodhurst, 1856.

Now, if, for a moment, we remove from consideration all those cases of rachitic origin which have been treated of indifferently by Grätzer and others as intra-uterine fractures, and confine our attention to those more notable cases of fracture where the bones have been healthy, we shall find that in every instance a severe mechanical injury has been received. Further, in almost every case of excessive congenital distortion with which I have met, some accident, involving violence and personal injury, has occurred during pregnancy. Fracture in utero and congenital distortion would seem, then, to have this in common, that physical injury has excited abnormal muscular action in the fœtus. If this be admitted, then every case of intra-uterine fracture and congenital distortion (whether the bones be healthy or ossification be most incomplete; whether fracture be partial, complete, or compound; whether distortion be slight or excessive) may be similarly explained, cause and effect varying, not alone, perhaps, as the cause varies, but as the effect of the injury is modified by temperament and other like condition.

The importance of this subject in a medico-legal point of view must plead my apology for the length at which I have stated my views in presenting these two cases to the Society. I must own that the more I reflect on the subject the closer and more striking does the analogy appear between fracture and distortion in the fœtus.

Malformation is frequently observed to exist together with fracture and with distortion. It must be considered merely as a coincidence, however, favouring in some instances fracture and in others distortion. It has its origin in the germ, and therefore is not essentially connected either with fracture or with distortion.

CASE OF
FEMORAL HERNIA,
WHICH HAD
DESCENDED BENEATH THE PECTINEAL PORTION OF THE FASCIA LATA,
AND WAS
PARTIALLY COVERED BY THE PECTINEUS MUSCLE.

BY

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Received Jan. 3d.—Read March 27th, 1860.

December 8th, 1859.—My attention was directed to the case of a female, æt. 45, who was admitted into the London Hospital, labouring under the usual symptoms of strangulation of the intestine, namely, constipation, vomiting, and pain in the abdomen. The history of the case was exceedingly obscure, but after the most careful inquiry of the patient herself, of the house-surgeon, and of a surgeon who had been called to see her, I came to the conclusion that, as no hernial tumour could be felt in the region of the crural ring, the case was one of femoral hernia, which had been reduced by the taxis, and in which the symptoms continued after the reduction of the hernia, probably from the effects of the stricture on the intestine. All that could be distinguished in the crural region were two moveable lymphatic glands, somewhat enlarged. The woman herself complained of some dull pain in this region.

Acting on the opinion I had thus been led to entertain, in which I was supported by my colleague, Mr. Ward, I ordered her to take a grain of opium every four hours. Under this treatment the sickness subsided, the pain diminished, and she was able to retain small quantities of tea, &c., upon her stomach. The third day after the commencement of this treatment, the symptoms reappeared with increased intensity: this induced me to make a further examination, but I was unsuccessful in discovering any swelling resembling in the least a femoral hernia. I now ordered her an injection of warm water, night and morning, to take a grain of calomel, with opium, thrice daily, and to have some wine and brandy, if they could be retained.

The symptoms still continued, the matter vomited became more stercoraceous in character, and on my visit on the 13th everything seemed changed for the worse.

I had previously explained to Mr. Ward my intention to adopt an explorative operation in the femoral region if the exacerbation of her symptoms justified a measure of this doubtful character. I again directed my attention to the femoral region, and I now fancied I could feel a fulness considerably to the inner side of the usual site of femoral hernia, and, as it appeared to me, under the edge of the adductor longus; two moveable, lymphatic glands were readily distinguishable over this apparent swelling. I therefore ordered her to be brought to the operating theatre, and having turned the thigh outwards, I was able to bring more fully into view the fulness to which I have alluded, and I felt at once amply justified in proceeding with the explorative operation.

The operation.—I divided the skin and superficial fascia in a vertical direction over the swelling, and by cautious dissection came down to a dense, elastic fascia, raised slightly into the form of a *swelling* rather than a *tumour*. In clearing this of its cellular investment I divided a vein, which I took to be the saphena, about to terminate in the femoral vein. I was obliged to tie it. With my finger I could clearly distinguish the falciform boundary of the saphenic opening in the fascia lata on the outer side. The glistening and tense fascia which was now exposed I regarded, from its position and depth, as the pubic portion of the fascia lata; I carefully scratched it, as it was exceedingly tense, and, passing a director beneath it, I divided it. I now distinguished, overlapping the swelling, some red fibres, which, from their direction, I thought to be a part of the pectineus muscle; I passed a director under the edge of this muscular band, and divided it transversely inwards. The hernial sac was at once apparent; I traced its neck upwards towards the femoral ring, and then opened it with the usual precautions; a small quantity of fetid pus at first escaped, and a portion of dark omentum, much congested, with about an inch and a half of small intestine, was exposed. The gut was obviously gangrenous, and a small quantity of fetid fluid oozed through some openings in it. Owing to the distance, I had some difficulty in reaching the seat of stricture, which was at the femoral ring; the stricture, unusually tight, was divided upwards and inwards, and immediately a considerable quantity of fæcoid matter escaped from the bowel through the wound. I cut off the omentum, dressed the parts lightly with lint soaked in warm water, and ordered an increased quantity of brandy, and as much liquid nourishment as her stomach would bear. She died four days after the operation.

Post-mortem examination.—I raised the skin and superficial fascia from Scarpa's triangle; and I also exposed the lower part of the tendon of the external oblique and Poupart's ligament. It was apparent that the hernia had descended through the femoral ring, not in the sheath of the femoral vessels, but behind and somewhat internal to the femoral vein, in the direction of the pectineus muscle; indeed, it was placed between the vein and the pectineus muscle. The rupture had deeply excavated the muscle, apparently by pressure upon it; but a few of the inner fasciculi, namely, those arising near the spine of the pubis and on a level with the adductor longus, projected in the form of a fleshy column, and had overlapped the hernia. It was this which I had cut transversely with my bistoury during the operation. The remainder of the muscle on which the hernia was situated was atrophied and somewhat sloughy. The strong column of muscle just mentioned was partly covered by the pectineal portion of fascia lata; but there was only a ragged vestige of this fascia covering the remainder of the atrophied pectineus. On sponging away the sloughs from the muscle, its surface was seen excavated into a deep cup-shaped depression, in which the hernia had been contained.

The hernia consisted of omentum and about an inch and a half of the middle of the small intestine. The omentum was firmly adherent to the neck of the sac, and appeared to be dragged down towards the femoral ring; the intestine contained in the sac was quite gangrenous, and the hernial sac itself was in a sloughy state. There was no great distension of the intestines above the hernia, but below this point the small intestines were empty. The peritoneal surface was inflamed, and covered by recently effused lymph.

There is some difficulty in coming to a very accurate conclusion as to the true nature of a case where the parts are in a state of gangrene, as in this instance, and where the coverings of the hernia are destroyed. But the observations made during the operation, and by dissection after death, lead to the following inferences: 1st, that the hernia was of the femoral species; 2dly, that the descent had not taken place in the sheath of the femoral vessels; 3dly, that it had passed behind this sheath, and had made its way behind and internal to the femoral vein, through some unusual opening in the pectineal portion of the fascia lata; 4thly, that that part of the pectineus muscle on which the hernia lay was deprived of its immediate fascial investment, and that the innermost fasciculus formed a strong fleshy column over the hernial sac, beneath the pectineal portion of fascia which covered it. In this respect the hernia differs from that variety of femoral hernia which descends in the sheath of the femoral vessels. Lastly, I believe that the hernia had existed for a long time as an *epiplocele*, and that the hernial symptoms arose from a sudden descent of small intestine behind the omentum.

CASE

OF

IMPERFORATE ARCH OF THE AORTA

IN WHICH

THE ROOT OF THE AORTA WAS RUPTURED.

BY

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Received Jan. 20th.—Read April 10th, 1860.

F. B—, æt. 24, employed at a wholesale druggist's, was admitted into St. Thomas's Hospital on December 8th, 1859. He stated, repeatedly and positively, that he had been well in all respects until the previous 21st of November; and his sister made the same statement, both at the time of his admission, and subsequently, when closely questioned, after his death. On that day—seventeen days before he was seen by me—whilst lacing his boot, he was suddenly attacked with very severe pain in the left side of the head, which, to use his own expression, almost immediately passed to the left arm and left side, and then across the lower half of the anterior part of the chest. He continued to suffer severely until four days before admission into the hospital. His breathing also became difficult; he had a sense of suffocation and palpitation; and was obliged to sit up in bed. His medical attendant told him he had disease of the heart; and also said, when all the distressing symptoms became greatly mitigated at the end of a fortnight, that he would get well in time.

I found him in bed, with the head and shoulders much raised, and he could not long bear to be in any other position. He breathed quickly, but did not seem much distressed. Healthy respiratory sounds could be heard over the whole chest, with the exception of the space about to be described, over which there was great dulness on percussion. The boundaries of this space were between the third and fourth ribs, extending from an inch to the right of the sternum, to near the line of the left nipple; a line drawn perpendicularly downwards on the left side, and a line curved a little outwards on the right, so that at one spot it was nearly two inches from the sternum, and the margins of the ribs. The heart's sounds over the ordinary cardiac region, and in all parts to the left of the sternum, were distant and muffled; and at the base a distinct systolic bruit could be heard. There was no impulse on the left of the sternum; and, until I had examined the dull space on the right side, I was under the impression that the pericardium was distended with fluid. When this space was examined I found the heart-sounds much louder and more distinct than elsewhere; and, in the spaces between the third and fourth and the fourth and fifth ribs, close to the right side of the sternum, pulsation could be seen and felt. The pulse at the wrist was very feeble.

I saw the man on the following day about two o'clock, when he said he felt better and more comfortable. No change had then taken place in the symptoms. At four o'clock he was observed to be very pale, and to struggle a little, and he died almost immediately.

Sectio cadaveris.—With the exception of a few old adhesions, and a little congestion of the bronchial glands, the larynx, trachea, bronchial tubes, lungs, and pleuræ were quite healthy.

The pericardium was much more in contact with the front of the chest than natural. On laying it open, it appeared, at first sight, to be filled with coagulum. On further examination, however, the entire pericardial surface was found to be lined by a thick, shaggy layer of recent, yet tolerably consistent lymph; the opposed surfaces of which were united, in many places, by adhesions. The intervals between these adhesions were occupied by a little bloody serum; and by an abundance of recently coagulated, black-currant-jelly-like blood, forming, in places, a layer three quarters of an inch thick. The greater part of this could be readily removed; but part was intimately entangled among the adhesions, so that, when the parietal pericardium was removed, the heart remained covered by a large quantity of lymph with entangled clot, and appeared much larger than natural. On removing this in part, it was seen that in the situation of the right auricle, bounded below by the auriculo-ventricular sulcus, and to the left by the great arteries, there was a rounded mass, about half the size of the fist, of what appeared to be solid coagulum, but separated from the pericardial effusion by the visceral pericardium. On opening the right ventricle, its walls were somewhat thickened and its cavity dilated. The right valves were healthy; but the tricuspid seemed to be much displaced backwards, by great expansion of the arch of muscular tissue separating it from the conus arteriosus; which arch evidently formed the inferior boundary of the mass of coagulum before alluded to, and was infiltrated with black blood up to the endocardial surface. The right auricle was evidently much encroached upon by the same mass of coagulum. The left ventricle was a little dilated and thickened. The mitral valves were healthy. That portion of the aortic orifice which is in a line with the lowest part of the valves was a little dilated; but from this point upwards, to the origin of the innominata, the aortic arch was dilated to the size of a goose's egg; the dilatation extending almost exclusively to the right and back, producing much of that encroachment on the right auricle which had appeared, at first, to be due solely to the clot. In consequence of the sudden and low commencement of the dilatation, the angles of the aortic valves had become widely separated; and the valves, consequently, expanded and attenuated. The anterior and left curtains were blended by congenital defect into one; a vertical frænum, which bisected the upper aspect, indicating its compound nature. The free edge of this measured two inches and a half in length. The right valve was much more attenuated than the others, presented no distinct corpus Aurantii, measured nearly nine tenths of an inch in depth, and two inches and a quarter in length along the free edge. The aortic valves seemed to be competent.

The dilatation of the aorta was chiefly behind, to the right and above the curtain last spoken of. The dilatation had a smoothly rounded form, and the walls of the ascending arch appeared smooth and healthy; and of normal thickness. There was no trace of atheroma.

At a distance varying between three quarters and half an inch above the compound aortic valve, was a nearly horizontal, irregular laceration, measuring an inch and a half in length, which, at its centre, gaped about half an inch. It extended from the orifice of the right coronary artery to below the orifice of the anterior one. A small horizontal rupture, about half an inch in length, was situated behind the first, and about half an inch from it. These ruptures were evidently of recent date, their margins presenting no deposit of lymph or other change; they opened, not into the pericardium or any of the cardiac cavities, but into the cardiac cellular tissue subjacent to them; and the blood had been forced through them into the cellular tissue and into the muscular substance of the anterior wall of the right auricle, and of that arch which separates the tricuspid and pulmonic valves—forming, in these situations, the large rounded mass of coagulum before spoken of. The effusion of blood extended, though in a lesser degree, into the cellular tissue and fat around the origins of the great vessels, and separating the auricles from the ventricles.

The sequence of events producing the conditions here described was probably the following:

1. Laceration of the aorta occurring at the time of the first sudden seizure.

2. Extravasation of blood into the substance of the heart.
3. Pericarditis.
4. Escape of blood into the pericardium in consequence of rupture of its visceral layer. This probably took place immediately before death. I had seen him an hour previously, and he was then decidedly more easy than when I first saw him, twenty-four hours earlier; and no change after this time was observed until a few moments before he died.

It has already been stated that the ascending arch was very much dilated; the dilatation being aneurismal in form; but smooth and uniform, and with perfectly healthy walls. The dilatation ceased an inch below the orifice of the innominata artery; and from this point to half an inch beyond the ductus arteriosus, it was not only smaller than natural, but gradually decreased in size; and, at the point indicated, presented a congenital constriction. There can be little doubt that the constriction had been complete; but, in removing the parts, the section had been made through the aorta immediately above the point of absolute obstruction—a small hole, sufficient to admit a probe, being left. It was ascertained that the lower part of the thoracic aorta was healthy, before the nature of the congenital malformation was detected; but then, unfortunately, it was too late to examine the state of the part at, and immediately beyond the point of complete obliteration. The ductus arteriosus formed a diverticulum from the aorta about as large as a goose's quill; and was pervious up to its nipple-like termination in the pulmonary artery. The innominata and the two sub-clavian arteries were much dilated; but the carotids were of their ordinary calibre. The internal mammaries were nearly as large as an ordinary writing quill. The obstruction of the aorta not having been noticed at the time when the examination was made, no further account can be given of the mode in which the circulation was carried on.

Although this man had been accustomed to hard work and to lift heavy weights, he had never suffered from dyspnœa, or shown any signs of heart disease, up to the period of his last attack. It is also worth noticing that there were no friction-sounds—indeed, scarcely any heart-sounds—although the pericardium was thickly coated with shaggy masses of lymph.

ON THE

TREATMENT OF AXILLARY ANEURISM.

BY

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Received March 17th.—Read April 24th, 1860.

THE operation for aneurism which constitutes the principal connecting link between the honoured name of John Hunter and practical surgery, has hitherto been regarded as one of the greatest improvements in surgical practice. The advantages of this procedure were indeed no less manifest than unquestionable with regard to the case for which it was proposed; since popliteal aneurism is in all respects unfavorably situated for the old operation, while the long stretch of undivided femoral artery affords free scope for acting on the new principle. Dr. Wilmer, of Coventry, tells us that, at the time he wrote (1780), there was not a single well-authenticated instance of this aneurism having been treated successfully by the old operation; and it is therefore not surprising that the facility and safety with which the disease was remedied by tying the artery in the fore part of the thigh, made a powerful impression upon all who became acquainted with this method of treatment. The old operation was consequently at once abandoned, and enterprising surgeons, in this as well as other countries, by their bold and successful efforts, opened a wider field for carrying out the Hunterian principle. Some traumatic aneurisms, more especially those of the smaller arteries, were deemed proper exceptions from the new system, but as the great leading principle of practice, it was universally admitted that the disease should be combated by ligature of the artery beyond the confines of the tumour. Such is the principle which, in common with other teachers of surgery, I have inculcated for more than thirty years, and of which I now feel it my duty to question the soundness.

In every aneurism, whether large or small, traumatic or spontaneous, the aperture of the artery is situated towards the centre of the tumour, so that a portion of the vessel, longer or shorter according to the size of the sac, is contained within its cavity, or rather embraced by its wall. Thus, in a very large aneurism ascending from below the groin nearly to the umbilicus, for which I tied the common iliac, the arterial opening was found to be only a little higher than Poupart's ligament; and it might always be expected to possess this relation to the cavity, when the process of formation is kept in mind. It has become a long-established and generally received principle in surgical pathology, that the extent of artery thus enclosed in the aneurism, or rather comprehended by its sac, is unsound, and unsuitable for ligature, while the vessel beyond the confines of the tumour is free from any participation in the disease. But, when this opinion is fairly examined, it will be found devoid of any good foundation; since the artery, though in some measure isolated from its neighbouring connexions by the effused blood, still retains the same relation as before to its sheath, and consequently, receiving a full supply of nourishment, is nowise unfitted for the process of obliteration by ligature. Indeed, when we trace the progress of an aneurism from its small beginning to a large size, it appears inconsistent with reason to consider the vessel sound so long as it can be felt beyond the tumour, and diseased so soon as it becomes overlapped or enclosed by the expanding sac. Whether the case be traumatic or spontaneous, there thus does not seem to be any reason for looking upon the portion of artery in question as unfit for being the subject of operative procedure. Under the influence of this consideration, I have treated all the aneurisms at the bend of the arm, resulting from wounds of the humeral artery though venesection, which have come under my care, amounting to ten in number, by opening the sac, and applying ligatures on both sides of the aperture. Proceeding upon the same principle, in a case of carotid aneurism caused by the thrust of a knife, at the lower third of the throat, and which extended from below the clavicle upwards, I did not scruple to lay open the cavity, and while hæmorrhage was restrained by pressure of my finger, to tie the vessel above as well as below the aperture, with the happy result of not only saving the patient's life, but also of preventing his assailant from forfeiting his, which under the circumstances he certainly would have done in the event of the case proving fatal. In a case of axillary aneurism, in a gentleman of about fifty-two years of age, where ligature of the subclavian was prevented by intense inflammation of the arm, rapidly running on to gangrene, I performed amputation at the shoulder-joint, cutting through the sloughy sides of the aneurism, and tying the artery where it lay within the sac. The patient made a speedy recovery, and lived in good health for several years afterwards. In another case of axillary aneurism, of which the subject was a gentleman of about twenty-two years of age, and in which also there seemed to be no alternative, I amputated at the shoulder-joint, with the same satisfactory result. But the following case, which lately occurred in my hospital practice, affords a much more striking illustration of the principle in view, and may, I think, not improbably suggest an alteration in the practice generally followed.

David Lockie, æt. 47, applied for admission into the hospital on the 26th of January, on account of a large tumour, which completely filled the left axilla, and greatly distended the muscles, before as well as behind the shoulder. The skin was tense, but not at all discoloured, and an obscure fluctuation could be perceived throughout the whole extent of the swelling, which the patient positively stated had not existed more than a week, although for nearly two months he had felt pain in his shoulder, and observed that there was no pulse at the wrist. There was a distinct aneurismal bruit, but no pulsation that could be felt in the tumour, except at its upper part, which projected above the clavicle. As in these circumstances there could be no hesitation in regarding the disease as an aneurism, careful inquiry was made as to the probable cause of its origin, but at first without success, since the patient positively denied having experienced any blow or wrench adequate to produce the effect. At length, however, he stated that, being tenant of a saw-mill, he occasionally assisted his men in turning heavy logs by means of the "cant-hook," and that upon such occasions—having had his left hand amputated at the wrist-joint by myself twelve years ago, on account of an injury from a saw—he was accustomed to rest his elbow on the pole, which was apt to spring up with great force, and thus subject his arm to a violent jerk at the shoulder. Having met with a case of axillary aneurism which was caused by the patient simply raising his arm to prevent his hat from being blown off as he got out of a railway carriage, I deemed this explanation sufficient.

In the course of a few days after admission the tumour enlarged considerably, and assumed a dark colour on some parts of its surface; while a slough formed over the scapula, where the pressure was most severe. At the same time the patient began to wander in his ideas, and his pulse rose to 130. It was therefore manifest, that unless some decided steps were taken without delay to afford relief, the result must very soon prove fatal. Ligature of the subclavian was quite out of the question, from the tumour extending above the clavicle; and amputation at the shoulder-joint at first seemed to be the only alternative. But before proceeding to this desperate remedy, I felt desirous of ascertaining the state of matters in the axilla, and therefore proceeded in the following manner.

On the 1st of February, chloroform having been administered, I made an incision along the outer edge of the sterno-mastoid muscle, through the platysma myoides and fascia of the neck, so as to allow a finger to be pushed down to the situation, where the subclavian artery issues from under the scalenus anticus and lies upon the first rib. I then opened the tumour, when a tremendous gush of blood showed that the artery was not effectually compressed; but while I plugged the aperture with my hand, Mr. Lister, who assisted me, by a slight movement of his finger, which had been thrust deeply under the upper edge of the tumour and through the clots contained in it, at length succeeded in getting command of the vessel. I then laid the cavity freely open, and with both hands scooped out nearly seven pounds of coagulated blood, as was ascertained by measurement. The axillary artery appeared to have been torn across, and as the lower orifice still bled freely, I tied it in the first instance. I next cut through the lesser pectoral muscle, close up to the clavicle, and holding the upper end of the vessel between my finger and thumb, passed an aneurism-needle, so as to apply a ligature about half an inch above the orifice. The extreme elevation of the clavicle, which rendered the artery so inaccessible from above, of course facilitated this procedure from below. Everything went on favorably afterwards. The edges of the wound, which had been brought together by stitches of the silver suture, united chiefly by the first intention. The ligature was found loose on the thirteenth day, the pulse gradually decreased in frequency as the patient regained his strength, and the discharge, which was at first profuse and mixed with clots of blood, progressively diminished. He was dismissed on the 14th of March, six weeks after the operation, and has since resumed his employment, in the possession of perfect health.

The circumstance in this case which appears to me most deserving of attention in a practical point of view, is the method that was employed to obtain command over the artery before it was tied, since, unless this had been done, it is obvious that a fatal hæmorrhage must have speedily ensued after the sac was opened. Thirty years ago I acted upon the same principle in my first operation for removing the superior maxillary bone. An attempt had been previously made by another person to accomplish this object, but without success, on account of the hæmorrhage; and, although at a loss to understand how it should prove troublesome, if the incisions were properly conducted, I resolved to obtain command over the only source from which the blood could proceed from the deep part of the wound, which was the internal maxillary artery. With this view I made an incision between the ramus of the jaw and mastoid process, so as to enable the gentleman who assisted me, the late Sir George Ballingall, to introduce his finger, and compress the vessel on the neck of the condyle. This had the desired effect, which, as I expected, proved quite unnecessary, and the operation was completed, for the first time in Great Britain, on the 15th of May, 1829.

I venture to hope that, through the employment of this expedient, axillary aneurisms not amenable to ligature of the subclavian artery may be remedied by the old operation; and I am inclined to think that, even in cases where the former procedure is practicable, the latter may be preferable. Upon two occasions I have tied the subclavian artery for aneurism with perfect success, and have therefore no prejudice against the operation; but, nevertheless, I feel satisfied that it is much more difficult of execution, more apt to be followed by hæmorrhage, as well as fatal inflammation of the deep textures, and less certain to afford relief, than the other method. For it by no means necessarily follows, that after the artery has been tied, the contents of the aneurism are always absorbed, while, when the sac is opened, the whole of the effused blood is at once removed.

How far the principle for which I now contend may be applied to aneurisms of the groin, requiring a high ligature of the artery, I shall not at present inquire. But if the practice should be so extended, those surgeons who prefer pressure and position to ligature for their treatment of popliteal and femoral aneurisms will find their field for performing the Hunterian operation so limited, that what has hitherto been held the rule of surgery, will be regarded as an exception.

POSTSCRIPT.

On the 15th of August, in accordance with the principles above explained, I performed the old operation for an aneurism, not traumatic, at the lower part of the axilla, in a gentleman about fifty, recommended to my care by Dr. Embleton, of Embleton, in Northumberland. The patient returned home on the 5th of September.

REPORT

ON

THE CONDITION OF THE PROSTATE IN OLD AGE,

FOUNDED ON

THE DISSECTION OF ONE HUNDRED SPECIMENS

IN

INDIVIDUALS OVER SIXTY YEARS OF AGE.

BY

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COMMUNICATED BY
HENRY THOMPSON, F.R.C.S.

Received Jan. 26th.—Read May 8th, 1860.

The difference of opinion as to the liability of the prostate gland to become enlarged in advanced life, has induced me to make some investigation into the actual condition of the part under these circumstances.

With this view, I have dissected the prostate in one hundred persons above the age of sixty; and I now have the honour to lay the result of my examinations before the Society. In order to facilitate the consideration of the cases, I have divided them into three classes, according to their weight.

We have thus, first, those under four drachms; second, those between four and six drachms; and third, those over six drachms.

My reason for adopting this arrangement, which at first sight may appear rather arbitrary, is, that we at once separate those glands which are most materially altered from those that are comparatively healthy.

It is evident that the weight of a prostate may generally be taken as a fair index to its condition, as to increase in bulk; and experience shows that glands weighing more than six drachms have become so altered as generally to inconvenience the patient. They must, therefore, be considered as hypertrophied, unless affected with some other disease.

Those between four and six drachms approach the normal size, which is given by Mr. Thompson,[1] one of the latest authorities on the subject, as four drachms thirty-eight grains; and as none of the individuals from whom they were taken had suffered during life any symptoms of urinary obstruction, we may fairly conclude that these organs did not exceed the natural proportions.

As to those under four drachms, which were evidently less than natural, it may be questioned whether, at any former time, they had been larger, and subsequently became atrophied; or whether the size they presented at death was their original condition.

I fear it will not be easy to settle this point: yet with the purpose of separating clearly what may be considered the normal from the abnormal, they have been arranged in a distinct class, and are as follows.

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[1] 'The Enlarged Prostate,' by Henry Thompson, F.R.C.S., 1858, p. 10.

CLASS I.

Prostates under four drachms.

	No.	Age.	Weight.			No.	Age.	Weight.	
			Drs.	Grs.				Drs.	Grs.
A	1	76	3	35		11	78	3	20
A	2	76	3	20		12	82	3	20
A	3	71	3	50		13	71	3	40
A	4	87	3	30		14	75	3	50
B	5	79	2	30		15	76	3	40
C	6	80	3	5		16	67	2	20
	7	67	3	20		17	70	3	20
	8	69	2	40		18	75	3	20
	9	85	2	40		19	83	2	5
	10	77	3	40		20	74	3	0

		Min.		Max.		Med.	
Which gives	Age	67		87		75·9	
		Drs.	Grs.	Drs.	Grs.	Drs.	Grs.
	Weight...	2	5	3	50	3	10

A. Cases in which commencing circumscribed tumours were found, four in number.
B. Abscess in both lateral lobes, the consequence of stricture, one case.
C. Posterior lobe enlarged, one case.

CLASS II.

Prostates between four and six drachms.

	No.	Age.	Weight.			No.	Age.	Weight.			No.	Age.	Weight.	
			Drs.	Grs.				Drs.	Grs.				Drs.	Grs.
A	1	81	5	0	B	16	80	4	20		31	81	4	20
A	2	82	5	5		17	67	5	40		32	76	4	10
A	3	68	5	50		18	78	5	0		33	64	4	0
A	4	78	5	30		19	76	4	0		34	81	5	0
A	5	82	5	0		20	76	4	40		35	84	4	40
A	6	71	5	30		21	66	5	20		36	82	4	50
A	7	69	5	0		22	84	5	0		37	75	5	15
A	8	87	5	10		23	85	4	30		38	60	5	0
A	9	85	4	30		24	74	4	40		39	73	4	30
A	10	81	6	0		25	94	5	0		40	71	5	30
A	11	71	4	30		26	75	4	30		41	73	5	0
A	12	76	5	30		27	61	4	20		42	68	4	0
C	13	82	5	50		28	81	5	20		43	70	4	0
C	14	85	5	30		29	76	5	30		44	68	5	0
B	15	72	4	30		30	84	5	20		45	77	6	0

		Min.		Max.		Med.	
Which gives..................	Age	60		94		76·2	
		Drs.	Grs.	Drs.	Grs.	Drs.	Grs.
	Weight...	4	0	6	0	4	57

A. Cases in which circumscribed tumours were observed, twelve in number.
B. Abscess existed in these two cases.
C. Slight enlargement of posterior lobe, two cases.

CLASS III.

Prostates over six drachms.

No.	Age.	Weight.		Part enlarged.	Nature of enlargement.
		Drs.	Grs.		
1	72	28	0	All lobes.	Tumours.
2	77	9	20	do.	do.
3	77	6	20	do.	do.
4	63	14	0	do.	do.
5	80	19	30	do.	do.
6	86	7	20	do.	do.
7	87	7	30	do.	do.
8	75	16	0	do.	do.
9	78	33	0	do.	do.
10	71	10	0	do.	do.
11	70	10	0	do.	do.
12	76	10	0	do.	do. [tumours.
13	72	17	0	do.	Fibrous hypertrophy, without
14	79	12	0	do.	do.
15	81	6	35	do.	Fibrous hypertrophy, with abscess.
16	79	30	0	do.	do.
17	76	24	0	do.	Tubercle.
18	78	30	0	Lateral lobes chiefly.	Tumours.
19	76	48	0	do.	do.
20	74	25	0	do.	do.
21	62	6	15	do.	do.
22	74	7	10	do.	do.
23	78	7	5	do.	do.
24	77	6	15	do.	do.
25	64	14	0	do.	do.
26	84	26	30	do.	do.
27	75	7	0	do.	do.
28	71	9	30	do.	do.
29	77	9	30	do.	do.
30	60	14	0	do.	Tumour, with abscess.
31	81	12	0	do. [only.	Fibrous hypertrophy, without tumours.
32	67	17	0	Left lateral & posterior lobes	do.
33	74	9	10	Right lateral lobe chiefly.	Tumours, with abscess.
34	81	8	30	Left lateral lobe only.	Fibrous hypertrophy, with abscess.
35	80	8	30	Posterior lobe chiefly.	Tumours.

Which gives		Min.		Max.		Med.	
	Age	60		87		75·2	
		Drs.	Grs.	Drs.	Grs.	Drs.	Grs.
	Weight...	6	15	48	0	15	2

Thus it appears that 35 per cent. of all prostates after the age of sixty are abnormally large; 20 per cent. are abnormally small; and 45 per cent. are within the limits of the normal weight.

It appears from the nearly equal average age in all three classes, that the condition of the prostate does not materially affect the longevity of the individual. A slight difference, however, does exist in favour of those in whom the gland is most nearly normal; the average in these being 76·2, and in the enlarged 75·2.

Nature of Enlargement.

This enlargement, which is found in 35 per cent. of all prostates after sixty years of age, is produced principally by hypertrophy of the fibrous tissue which naturally exists in the organ. The gland-follicles also become enlarged, and more numerous, but do not affect the size of the organ to the same extent as the fibrous development.

This new tissue may be found deposited equally throughout the organ, which in that case presents on section a smooth and even surface; but most generally it is seen to be exaggerated at certain parts, producing, in some instances, tumours, which start out from the surface of a section and make it irregular. Between these two conditions there are many gradations; the fibrous tissue may be seen lying in concentric layers of greater or less size, and more or less distinctly marked; giving the appearance as if, by their increase and progressive hardening, they gradually led to the formation of tumours.

Their minute characters and mode of formation have already been so well described by different authors, that they need not be entered into here.

The frequency of this fibrous deposit is shown by the fact, that of the thirty-five cases of enlargement it existed in thirty-four. In seven of these there was no appearance of tumours; in twenty-seven they were present in various degrees of development.

It may be remarked that these tumours are not peculiar to the enlarged condition of the gland, for they were found in four of the abnormally small glands and in twelve of the normal; so that they exist in 43 per cent. of all prostates after sixty years of age, and we may reasonably conclude that their presence in the smaller prostates indicates a tendency to enlarge, which, with a few years' longer life to the patient, might have become developed, and have caused him serious inconvenience.

With regard to the tumours found in the smaller glands, those between four and six drachms and under four drachms, it must be understood that they were not distinctly marked, or capable of being readily separated from the surrounding tissue, as was frequently the case in those glands where they had attained a greater development; but they appeared to be only in the incipient stage: that is, at one or more particular spots, the fibrous tissue was seen to have become hardened, probably from additions to its material, having a firm feel on pressure, appearing as if it had contracted upon the included softer tissue, and giving a slightly nodulated appearance to a section. These appearances may be gradually traced, becoming more and more marked as the development of these tumours advances, or as further additions to the fibrous tissue are deposited.

Seat of Enlargement.

It is of importance to determine which part of the prostate is most liable to enlargement, as the flow of the urine is much more likely to be obstructed by enlargement of one portion than of another.

From the peculiar relation of the posterior lobe to the mouth of the bladder, it is easily understood how the projection of it, when enlarged, more seriously obstructs the passage of the urethra than when the lateral lobes alone are increased in size. The relative frequency of enlargement of these parts, in the thirty-five enlarged glands, will be observed in the following table:

Table showing the frequency of enlargement in different parts in thirty-five cases.

In	17	enlargement	existed	in all three lobes, pretty equally.
„	14	„	„	in both lateral lobes chiefly.
„	1	„	„	in left lateral and posterior lobe.
„	1	„	„	in right lateral lobe mostly.
„	1	„	„	in left lateral lobe alone.
„	1	„	„	in posterior lobe chiefly.

Thus it appears that enlargement of the posterior lobe is not of so frequent occurrence, comparatively with increase in other parts of the organ, as was at one time supposed. In a little more than a half of all enlarged glands, it is found, either associated with enlargement of the other parts, or singly—by far most frequently the former; and among the sixty-five glands under six drachms weight, only one case occurred in which this portion had become developed to a degree that might be supposed capable of opposing the flow of urine. We have thus 20 per cent. of all prostates over sixty years of age, in which the posterior lobe forms an obstruction at the mouth of the bladder.

The occurrence of only one case throughout the whole series, in which the posterior lobe alone was enlarged so as to cause obstruction, the other parts being healthy, shows the rarity of that condition of the prostate.

The particulars of this case are perhaps worth mentioning. It is No. 6 in Class I. The gland was considerably under the average size, weighing three drachms five grains. No irregularity of shape was noticeable till the bladder was opened, when the posterior lobe was found elongated, and rising as a valve over the mouth of the urethra, so as, when applied, to close it almost completely.

That it had caused obstruction to the escape of urine, although the patient never had been under treatment for such symptoms, was evident from the fasciculated state of the bladder, while the urethra itself was free from stricture. It may be doubted whether this projection should be considered a true hypertrophy of the posterior lobe; for it seemed to be not more intimately connected with that part of the prostate, than with a large fasciculus of the muscular coat of the bladder, which passed from the mouth of the urethra, along either side of the trigone; but wherever it originated, it effectually acted the part of an enlarged posterior lobe, and therefore I have classed it with these cases.

Serious opposition to the flow of urine may also arise from enlargement of the lateral lobes, especially when tumours exist, and lie towards the centre of the organ; as then they may project, and encroach upon the canal of the urethra, distorting it to the right or left, and narrowing the passage to a mere chink, while at the same time its extent of surface is greatly increased. The obstruction from this cause, however, although impeding the escape of urine, is not likely ever to do so to the same degree as enlargement of the posterior lobe; nor does it present so serious an obstacle to the passage of an instrument into the bladder.

It may be remarked that, in cases where the hypertrophy is advanced, and the tumours tend inwards, the urethra becomes greatly expanded, and the interspaces between the rounded sides of the projections into it, serve as channels for the urine to pass away by. This condition will frequently explain, I have no doubt, the occasional absence of symptoms of obstruction in cases where the prostate is known to be considerably enlarged.

Frequency of Symptoms.

The most important symptom indicating the presence of enlarged prostate, and, at the same time, the most direct result of the condition of the parts, is retention of urine, either complete or partial. It is of consequence to know, however, that this symptom does not present itself in every case of enlargement.

The proportion of men in advanced years suffering from the consequences of enlarged prostate, is indeed small. Thus, amongst 1600 old men, with an average sick-list of 200, not more than ten are under treatment for this disease; and half of these only occasionally. A much larger number must be affected with enlargement of the gland, as shown by the results of post-mortem examination; but, from a variety of causes, although in several probably symptoms were present, they did not seek medical aid. Of the thirty-five cases of enlargement, fifteen made no complaint to the medical officer during life; with the exception of three in whom symptoms appeared after lesions of the nervous system. The following table shows the nature and extent of the alteration in the glands, in these cases:

Table showing the nature and extent of alteration in fifteen cases of enlargement, without symptoms.

No.	Age.	Weight.		Part affected, and how.
		Drs.	Grs.	
1	80	8	30	Posterior lobe chiefly; lateral lobes slightly; tumours.
2	81	8	30	All lobes; tumours; abscess of left lateral lobe.
*3	80	19	30	All lobes; tumours; apoplexy.
4	62	6	15	Lateral lobes; tumours.
5	74	7	10	Ditto ditto.
6	78	7	5	Ditto ditto.
7	77	6	15	Ditto ditto.
8	86	7	20	Ditto ditto.
*9	64	14	0	Ditto ditto; softening of brain.
10	84	26	30	All lobes; tumours.
11	87	7	30	Ditto ditto.
12	75	7	0	Lateral lobes; tumours.
13	71	10	0	All lobes; tumours.
14	71	9	30	Lateral lobes; tumours.
*15	79	12	0	Posterior lobe chiefly; lateral lobes less so; tumours; apoplexy.

* Symptoms, in these cases, appeared after lesions of the nervous system.

Although many of these cases were not greatly enlarged, some of them, as Nos. 1, 3, and 10, plainly showed that the prostate may be very greatly altered and enlarged, and yet the patient be free from urinary obstruction.

Case 1, æt. 80, died of pneumonia, and never suffered symptoms of urinary obstruction. The prostate weighed eight drachms thirty grains. The posterior lobe was prominently enlarged, projecting in a triangular shape, and covering the mouth of the bladder.

Case 3, æt. 80, died of apoplexy two months after the attack, and had not previously suffered urinary symptoms. The prostate weighed nineteen drachms thirty grains, and was hypertrophied in all its parts. Tumours were abundant, and encroached on the urethra, especially on the left side, from which a pyriform growth arose, near the veru montanum: passing backwards, its rounded extremity projected through the mouth of the bladder. A similar growth, but of less size, existed on the right side.

Case 10, æt. 84, died of pneumonia, and had no urinary symptoms. The prostate weighed twenty-six drachms thirty grains. The enlargement was principally seated in the lateral lobes, but the posterior also projected slightly. Tumours were abundantly developed.

Case 3 also shows, what is commonly remarked, that the enlarged condition of the gland may exist unnoticed until the powers of the system are broken down, and the bladder paralysed from some nervous lesion. Then the expelling force is impaired, and the unnatural obstruction discovers itself.

OTHER CONDITIONS.

Concretions.

The other alterations of the prostate besides enlargement, which were noticed in these dissections, were comparatively rare, with one exception. I refer to the presence of the small black concretions; and perhaps this ought not to be considered as an alteration, as in every case where they were looked for they could be discovered, either by the unaided eye or by the microscope. Nor could any special condition of the organ be connected with their presence, as they were found in glands of all sizes, and in every variety of alteration. I am therefore disposed to consider them as normal to the gland in advanced life, rather than as a diseased condition in any way affecting the patient.

Abscess.

Abscess in the prostate appears rather as a consequence of obstruction to the flow of urine than as a disease likely to produce that condition. The cases I have met with seemed to arise from such causes as stricture of the urethra, obstruction at the mouth of the bladder from enlarged third lobe, or paralysis of the bladder; all tending to produce retention of urine, with its usual accompaniment of inflammation of the mucous surface of the bladder. The irritation seemed to have extended from this, and to have produced the suppuration in the prostate.

The frequency of abscess in the enlarged gland is in the proportion of five in thirty-five; in the non-enlarged, two in sixty-five.

The principal facts connected with these cases may perhaps be best seen by the following table:

Cases of Abscess of Prostate.

No.	Age.	Weight.		Probable cause.	Position of abscess.
		Drs.	Grs.		
1	79	2	30	Stricture of urethra.	Lateral lobes.
2	74	9	10	ditto.	Right lateral lobe.
3	60	14	0	ditto.	Posterior lobe.
4	81	6	35	Enlarged posterior lobe.	Posterior lobe.
5	81	8	30	ditto.	Left lateral lobe.
6	79	30	0	Enlarged posterior lobe, combined with paralysis of bladder.	Posterior lobe.
7	80	4	20	Paralysis of bladder.	Right lateral lobe.

Abscess in the prostate does not generally attain any great size. Although both lateral lobes are sometimes found quite excavated by suppuration, yet the capsule of the prostate, and the structure of the gland itself, offer such opposition to extension, that the pus generally soon makes its way into the urethra, by one or more openings, most frequently in the neighbourhood of the veru montanum.

Tubercle.

Tubercle is the only other abnormal affection of the prostate, giving rise to enlargement, which I have noticed; and that only in one case. The patient was aged seventy-six. The gland was affected chiefly at its circumference, the parts around the urethra being comparatively healthy. The neighbouring cellular tissue and the vesiculæ seminales were involved, producing a large tumour between the rectum and bladder; the prostatic portion of which, when removed, weighed about twenty-four drachms. A similar deposit was found in the lungs, in the right kidney, and in the mucous membrane of the bladder. This case is No. 17 of Class III.

Calculi.

It may be interesting, in conclusion, to note the occurrence of urinary calculi in connection with enlargement of the prostate.

In three cases, Nos. 3, 4, and 23 of Class III, urinary calculi were discovered in the bladder, on dissection. In Case 3 two uric-acid calculi, each about half a drachm in weight, were found. Great obstruction existed at the mouth of the bladder, and the man died suddenly, from rupture of the coronary artery of the heart, while making violent straining efforts to void water.

In Case 4 two phosphatic concretions were found; one weighed seven drachms forty-five grains; the other, thirty-five grains. A curious provision existed, in this case, for the protection of the mouth of the bladder, consisting of a projection from either lateral lobe of the prostate, close to the opening of the urethra; the two swellings served as buttresses, to defend the orifice from the calculus, and, at the same time, kept it free for the passage of urine.

In Case 23 one small phosphatic calculus was found, apparently of comparatively recent formation.

When we consider the favorable circumstances for the formation of phosphatic concretions in cases of enlargement of the prostate, where a pouch exists below the level of the mouth of the bladder, which can rarely be completely emptied, and must therefore favour the accumulation of sedimentary matters, it is surprising that calculi are not more frequently found.

ON THE

CONDITION OF THE BLOOD IN MANIA.

BY

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Received Feb. 7th.—Read May 8th, 1860.

During the last four years the attention of many scientific investigators has been directed to the condition of the blood in disease, and the discovery of some very valuable facts has been the result. In gout, for example, Dr. Garrod has shown most conclusively that uric acid is present in very considerable quantity. In certain altered states of the spleen and of the lymphatic glands, Drs. Virchow and Bennett have detected a great increase in the number of white corpuscles; and very recently Dr. Gübler has shown that there is a marked increase of the same white corpuscles during the last stages of cachectic diseases generally. Facts have accumulated indeed, among which there are instances which furnish support to the belief that there is some equivalent change in the blood in all diseases—some change which may give a key to a system of rational treatment; and that the blood in acute mania forms no exception to the general rule may be gathered from the investigations of Drs. Hittorf and Erlenmeyer.

Much remains to be done, however, before we can boast of having any exact knowledge of the state of the blood in insanity, and hence the author, with the able assistance of his friend Dr. Marcet, has been induced to seek for some new facts. These facts he has found in a careful analysis of the blood of six maniacal patients during the paroxysm and in a state of convalescence. The author is also duly sensible that much still remains to be done, but at the same time he thinks that the facts already arrived at show clearly (what has not been indicated before) *that there is a marked deficiency of fibrine during the period of maniacal excitement and a correction of this deficiency during convalescence;* and on this account he begs to submit the following remarks to the notice of the Fellows of the Royal Medical and Chirurgical Society.

Narrative of the Cases and Experiments.

The six cases selected for experiment were three of *acute* and three of *recurrent* mania. An outline of each case, sufficient to show the nature of the attack, with the treatment prescribed, is given, preceded by the chemical analyses of the blood made during the period of maniacal excitement, and afterwards.

Cases of Acute Mania.

Case 1.—W. G—, æt. 21. Admitted into Bethlehem Hospital in December, 1856. Was previously a draper's assistant. He became maniacal one month before admission. Hereditary tendency was traceable in both his father and paternal grandfather. No known exciting cause. He talked incessantly in a rambling, incoherent manner; was irritable and occasionally violent, at which times he would do injury to others, if not prevented. There was no apparent bodily disease. He was of spare habit, and an excitable temperament. Previous to his admission, he had not been subject to medical treatment. Nourishing diet was prescribed for him, and the following sedative mixture:

Morphiæ Acetatis, gr. ss;
Tinct. Hyoscyam., ʒj;
Mist. Camph., ℥iss; ter in die sumend.

At the end of a fortnight the morphia was increased to one grain three times a day, and he continued to take it for many months, but without any apparent benefit. In July his maniacal symptoms abated, and he improved gradually, until September, 1857 (nine months after admission), when he was discharged, perfectly cured.

Analysis of the Blood.

During the period of the highest mental excitement.		During convalescence, four weeks before leaving the hospital.	
Water	777·39	Water	784·93
Red particles	149·74	Red particles	110·07
Fibrine	1·74	Fibrine	2·20
Albumen and extractive matters	63·28	Albumen and extractive matters	90·63
Inorganic salts	7·85	Inorganic salts	8·00
		Fatty matters	4·17
	1000·00		1000·00

Remarks.—In the analysis made during the maniacal stage, the proportion of every constituent was determined by actual experiment, except the blood-corpuscles, which it was not possible to obtain otherwise than by calculation. The want of consistence in the *fibrinous clot* was remarkable, and the corpuscles were not at all retained in the network of fibrine, a result which was fully borne out by the chemical analysis. Nothing abnormal could be detected by the microscope.

In the second analysis (taken during the convalescent period), the sample of blood was peculiar, from the circumstance that the serum had a milky-white appearance. The microscopical examination failed to show the presence of fatty globules or of other abnormal anatomical elements. Chemical analysis indicated the existence of a rather large proportion of fatty matters; the *fibrine was increased* to its normal character, but the red particles were below the average. The *albumen* was rather high, and the *inorganic salts* somewhat deficient.

Case 2.—W. T. G—, æt. 33, a drum-major in the Guards, admitted into Bethlehem Hospital in March, 1857; was married, and had lived a steady, temperate life. Strongly built, of a muscular frame, a sanguineous temperament, and shy, retiring habits. Maniacal symptoms first appeared four months before admission, when he underwent medical treatment of an antiphlogistic character in the military hospital. No hereditary tendency could be traced, and the disease appeared to be solely referable to intense study, and an ambitious desire to make himself an accomplished musician. He had many delusions, principally of an exalted and extravagant character. He described himself sometimes as the "*Son of God*," at other times as an "*Earthly Sovereign.*" He was noisy, mischievous, and incessantly talking.

Morphia and sedatives, with full diet, were prescribed.

In February, 1858, he was so far convalescent as to be allowed to leave the hospital on a month's trial. At the expiration of the month he presented himself again, and was then finally discharged as sane.

Analysis of the Blood.

During the maniacal symptoms, in August, 1857.	
Water	791·64
Red particles	125·48
Fibrine	1·55
Albumen and extractive matters	69·84
Inorganic salts	8·98
Fatty matters	2·51
	1000·00

At the time of his first discharge.	
Water	758·76
Red particles	140·61
Fibrine	2·75
Albumen and extractive matters	87·28
Inorganic salts	8·82
Fatty matters	1·78
	1000·00

Remarks.—In this case, during the maniacal state there was a remarkable excess of *water*, and a deficiency of *albumen* and *extractive matters.* This increase of water was expected at the commencement of the analysis, from the slow coagulation of the blood during the process of drying in the water bath. The *red particles, fatty matters,* and *inorganic salts* were in a more normal proportion; the quantity of fibrine was deficient, being little more than one half its due proportion; the *serum* was clear, and there was no indication of excess of fat.

In the analysis made during the convalescent stage, the *serum* was perfectly transparent; it had a slight pink tinge, and was of the natural consistence.

The *clot* was normal. The proportion of *albumen* and *blood-corpuscles* was high, that of *water* low; the weight of fibrine normal, but the *fat* much below the average. It is remarkable that this low proportion of fatty matter was attended with a clear *serum.* The *ashes,* or *inorganic salts,* were in normal proportions; they exhibited a very red colour, probably from their containing a large quantity of peroxide of iron.

CASE 3.—E. R—, married, and the mother of four children, admitted into Bethlehem Hospital in June, 1857. She was of a sanguineous temperament, strong passions, and the subject of great sexual excitement, with direct hereditary tendency to mania on the mother's side. She had had three previous attacks since the age of 24, occurring on each occasion during a state of great debility. In the present instance the disease at the time of her admission had lasted one week. For some weeks after admission she was in a sullen and inert condition, seldom speaking to any one, and violent when opposed. She was very dirty in her habits and offensive in her conduct.

The case was treated for several weeks with half-grain doses of tartarized antimony three times daily. This produced no relief, but was followed by rather an aggravation of the symptoms.

She became excited and mischievous, used very obscene and profane language, and was difficult to manage. Morphia was substituted for the tartarized antimony.

In about eight months the symptoms of insanity suddenly disappeared, and she became cleanly and industrious, cheerful and well behaved. She was discharged quite well at the end of May, 1858.

Analysis of the Blood.

During the excited stage, in December.		On the day of discharge.	
Water	806·71	Water	800·90
Red particles . . .	104·68	Red particles . . .	109·32
Fibrine	1·67	Fibrine	1·75
Albumen and extractive matters . . .	76·53	Albumen and extractive matters . . .	74·99
Inorganic salts . .	7·41	Inorganic salts . .	8·89
Fatty matters . .	3·00	Fatty matters . .	4·15
	1000·00		1000·00

Remarks.—In the analysis during the *maniacal stage* the *serum* was milky; the *clot* of natural size and consistence; the proportion of *water* high, and the other constituents proportionably low; the *fatty matters* rather above the normal quantity, which might have been expected from the milky appearance of the serum; the fibrine below the average; the *albumen* and *red particles* under the normal proportion.

In the second analysis, when *convalescence* had taken place, the serum was very turbid, and of a nearly white colour. The proportion of *water* high, and a corresponding deficiency in the weight of the *red particles* and *fibrine.* An increase of the fatty matters found actually to exist, as anticipated from the state of the serum; the proportion of *inorganic salts* was normal, and that of the *albumen* and *extractive matters* rather low.

Cases of Recurrent Mania.

Case 4.—E. I. G—, æt. 58, a single woman, admitted into Bethlehem Hospital in 1837. When first attacked with mania, the disease was said to be consequent upon extreme grief on account of the death of her father. In this, as in many cases, the disease declared itself by a perversion of her usual habits, demeanour, and conversation. Her ideas became disordered; her language was extremely obscene. She destroyed her clothes, defied all authority, was violent and dangerous. It was necessary to place her in an asylum; and since that time she has been subject to paroxysmal attacks, which are of a most revolting character. Feuchtersleben gives a German physician credit for graphically comparing the movements of a maniac to those of a hyæna confined in a cage, and no untamed creature in the brute creation presents a more deplorable picture than this poor woman during her maniacal paroxysm. She distorts her face with the most hideous grimaces. She utters little, but that little in a growling, inarticulate manner; and her ejaculations and shrieks are of the most revolting character. In this wretched condition she remains during the day, sitting or crouching on the floor, for five consecutive weeks.

The maniacal symptoms then abate, and are followed by a state of composure and perfect sanity, the voice returns to its natural gentle tone, and when she becomes convalescent she is a diligent needle-worker, and her manners are feminine and gentle.

This happy change only lasts a fortnight, at the expiration of which period it is surely followed by a return of the maniacal paroxysm.

The uniform periodicity of these changes is not more painfully interesting to notice than the unvaried similarity of each attack. No excitement appears to hasten the hour of approach. No medical, moral, or hygienic measures hinder its occurrence or rob it of an hour's duration. All has been tried, but so certain as the fortnight of peace and mental purity has expired, so surely will the nurse, when the bed-room door is opened in the morning, find the patient out of bed, and lying on the floor in a state of maniacal frenzy.

The patient did not in either attack take medicines, nor was there any alteration made in the diet.

Analysis of the Blood.

During the maniacal stage, May 1st, 1857.	
Water	796·66
Red particles	121·77
Fibrine	1·58
Albumen and extractive matters	98·44
Inorganic salts	8·55
	1000·00

Convalescent stage, Nov. 11th, 1857.	
Water	811·68
Red particles	126·01
Fibrine	2·88
Albumen and extractive matters	47·54
Inorganic salts	9·31
Fatty matters	2·58
	1000·00

Remarks.—The first analysis shows that the *albumen, red particles,* and *ashes* were not far from the normal proportion. The *fibrine* was decidedly less than in healthy blood.

In the second analysis, at the convalescent period, the blood was found very watery, an excess of *inorganic salts,* the *albumen* and *extractive matters* greatly under the average amount in health. The proportion of *fat* and *red particles* were normal. The *serum* clear but slightly pink, the *clot* bulky, the proportion of *fibrine large.*

Case 5.—F. B—, admitted into Bethlehem Hospital in April, 1838, at the age of 27. The mental disease in this case was clearly traceable to hereditary tendency. The mother, who died insane, left five children, four of whom have laboured under the same disorder. F. B— is of an amiable disposition, but living in constant alternations of mental excitement and tranquillity. During the maniacal period she is violent, very obscene and insolent in her conduct, her language and actions bearing a strong impress of sensuality, and her habits dirty and disagreeable. In this state her usual attitude is one of sullen, determined resistance.

There is no fixed regularity in the continuance of the attacks, but whether extending over a shorter or longer period, are followed by seasons of tranquillity, depression, and amiability, her mind regaining its reasoning powers, and her conduct becoming mild and irreproachable. This state of mental alternation has continued for twenty years without any perceptible variation.

Analysis of the Blood.

Maniacal stage, July 16th, 1857.		Convalescent stage, August 11th, 1858.	
Water	784·07	Water	765·73
Red particles	123·81	Red particles	139 05
Fibrine	0·06	Fibrine	2·46
Albumen and extractive matters	81 69	Albumen and extractive matters	81·75
Inorganic salts	8·62	Inorganic salts	8 09
Fatty matters	1·75	Fatty matters	2·92
	1000·00		1000 00

Remarks.—The proportions of the various constituents of the blood were normal, except the *fibrine,* hardly any traces of which were obtained; that which was secured was of a gelatinous consistence.

In the analysis taken during the convalescent stage, the *serum* was clear, but rather darker coloured than usual; the *clot* was very firm and bulky. The *colouring matter* slightly in excess, *fibrine* normal, and the *fatty matters* in less proportion than usual.

Case 6.—W. D—, admitted into Bethlehem Hospital in June, 1841, at the age of twenty-nine years, of easy, amiable disposition, and good education.

Expression of countenance varying with the recurrent character of the mental disease; at one time bright and intelligent, at other seasons heavy, repugnant, and almost animal. He was formerly a mercantile clerk. His mental disorder was caused by the grief consequent upon the death of his father. For many years past his condition and delusions have assumed a recurrent periodicity; for a month or six weeks he will be found rational and conversable; during the five succeeding weeks his entire mental condition appears to have undergone a revolution, irritability and passion succeed the amiability, and he has the habits and bearing of a congenital idiot. His conduct is humiliating to witness, oftentimes repulsive, and his personal appearance seems to sympathise with this sad revulsion by assuming even a degraded and animal character. He passes the greater part of the day standing by his accustomed chair at the table, with plate and mug in hand, steadfastly eying the last meal that has been sent to him, and which he obstinately refuses to commence until the succeeding one has been almost finished by his companions. In this routine, he may be found beginning his breakfast as others are leaving the dinner-table, and eating his dinner when his associates are at their evening meal. He smells each piece, and subjects it to strict scrutiny before placing it in his mouth; and thus his day is passed. When convalescent, he willingly takes exercise, seeks pleasure and amusement in reading, playing bagatelle, or draughts, and though never a very lively companion, yet he is sensible and can be agreeable.

The analysis of the blood was taken at such periods as would best represent a fair specimen of each particular state.

Analysis of the Blood.

Maniacal stage, March, 1858.		Convalescent stage, July, 1858.	
Water	773·86	Water	779·93
Red particles . . .	135·56	Red particles . . .	121·65
Fibrine	1·96	Fibrine	2·97
Albumen and extractive matters . . .	79·22	Albumen and extractive matters . . .	87·16
Inorganic salts . .	7·99	Inorganic salts . .	8·29
Fatty matters . . .	1·41	Fatty matters . .	0·00
	1000·00		1000·00

In the analysis, during the *maniacal stage,* the serum was perfectly clear, and exhibited a pale-yellowish colour, with an alkaline reaction; the *clot* firm and bulky, had seized upon nearly all the corpuscles; a slight deficiency of *albumen* and *extractive matters,* and the proportion of *water* high. The fibrine was under the normal proportion, and the *fats* decidedly below the average; the *inorganic salts* rather low, but not abnormal.

In the *convalescent stage,* the *serum* was slightly hazy, and easily separated from the clot, which was very bulky; the proportion of *fibrine* was normal.

On comparing these cases with those of an analogous character which have been put on record by Drs. Hittorf and Erlenmeyer, the lesson taught by them becomes more pointed and cogent.

The following is the process by which each analysis was obtained:

The blood was drawn by cupping at the nape of the neck, and immediately collected in two small earthenware pots, containing about two ounces each.

Each analysis was conducted by precisely the same rules and according to the same method, and in each case about the same quantity of blood was extracted.

The analysis was commenced within a few hours of the extraction of the blood, and thus any loss of water, especially in warm weather, was prevented.

In each examination, the blood of one jar was first submitted to analysis for the determination of the albumen, extractive matters, and fibrine; the blood of the other jar being preserved for the determination of the water, globules, fatty matters, and inorganic salts.

In some cases the serum was clear; in others more or less milky; in others slightly tinged with blood-corpuscles.

The jar and serum having been previously weighed, a sample of serum was decanted into a weighed capsule, and the weight of it and its contents immediately ascertained. This serum was then evaporated to dryness in a water bath at 212° Fahr., the operation being continued until the serum had ceased to lose weight, and care being taken to leave the capsule for a short time in the air-pump vacuum over sulphuric acid previous to weighing it. The blood remaining in the jar (after the serum had been extracted from it, and consisting mostly of the clot) was transferred to a piece of white calico, the corners of which being secured in the shape of a bag, allowed the clot to be thoroughly washed in water, in order to separate the fibrine from the other constituents of the clot. As soon as the fibrine had become colourless it was carefully removed from the cloth by a pair of forceps into a small weighed capsule, and dried in a water bath at 212° Fahr. until it ceased to lose weight. The weight of the empty jar was finally ascertained.

The second jar having also been weighed, its contents were then transferred to a weighed capsule, and evaporated in a water bath at 212° Fahr., until the blood had ceased to lose weight, the same precaution being taken as in the case of the serum to leave the dry residue for a short time in the air-pump vacuum over sulphuric acid previous to weighing it.

The weight of the empty jar was also ascertained. From the dry blood was obtained the weight of the fatty matters and inorganic salts. For this purpose, the whole of the dry blood was pounded, in order to effect a mixture of the dry clot and dry serum, and then a sample was pounded still more finely and dried again (to remove the water it had absorbed during pounding), weighed, and then mixed with sulphuric ether, which dissolved out the fats contained in the finely pounded quantity of the dry blood. The ethereal solution, evaporated to dryness in a weighed capsule, yielded the proportion of fats in the finely pounded dried blood. A sample of the roughly pounded dry blood was placed in a weighed platinum capsule (at the time the blood had been ascertained to be perfectly dry), weighed, and burnt on a small gas flame, the burning being continued until the ashes had ceased to lose weight; thus the proportion of ashes in the dry blood which had been burnt was determined.

From these several data the composition of the blood was calculated.

M. Hittorf has recorded seven cases of acute mania, in which he analysed the blood during the stage of maniacal excitement; his results are the following:

Table of analysis of seven cases of Acute Mania.

Analyses of 1000 parts of blood.	FEMALE.				MALE.		
	Case 1. Girl, æt. 20.	Case 2. Girl, æt. 23.	Case 3. Married woman, æt. 37.	Case 4. Unmarried woman, æt. 30.	Case 5. Man, æt. 30.	Case 6. Man, æt. 47; periodical mania.	Case 7. Man, æt. 48.
Water	749·559	803·884	805·701	803·345	788·999	779·093	751·995
Blood-globules . . .	109·191	116·967	119·576	112·010	118·199	137·898	130·808
Fibrine	2·083	1·932	3·173	1·929	1·396	1·870	1·455
Albumen	83·913	68·590	65·099	74·324	79·577	73·353	75·042
Extractive matters and soluble salts . . .	7·786	7·980	6·075	6·114	8·603	7·432	10·106
Fats	2·468	0·647	0·376	2·278	3·226	0·354	0·594

Dr. Hittorf concludes, from these cases, that in acute mania there is a diminution in the amount of the globules, and an increase in the amount of water; but he does not believe that the blood undergoes such a change as to lead *à priori* to the assumption of the fact. We, however, consider that these cases show more than this, and that what they do show is in harmony with our own observation, for in six out of the seven cases, is there not *a distinct diminution in the amount of the fibrine of the blood?*

M. Erlenmeyer, of the establishment for the insane at Prague, states that he has made 304 analyses of the blood, but principally in cases associated with other internal diseases. He only details three cases, and in two of them mania was accompanied by epilepsy. The blood was drawn during the paroxysm.

	Case 1. Male, æt. 66; violent mania, recurrent.	Case 2. Male, æt. 22; mania and epilepsy.	Case 3. Male, æt. 34; mania and epilepsy.
Water	798·937	815·747	803·242
Blood-globules . . .	114·126	105·596	118·544
Fibrine	1·853	2·301	1·721
Albumen	73·635	65·830	67·325
Salts and extractive matters .	10·820	9·811	8·530
Fat	0·629	0·715	0·638

The conclusions drawn by Dr. Erlenmeyer from these cases, and from others which are not quoted, is, that augmentation in the number of red particles, or in the amount of fibrine, is a very unusual occurrence in insanity; and that the opposite state, *i. e.* diminution of the red particles and increase of serum, is not an unfrequent condition. In two of the three cases given, the *amount of fibrine is deficient,* but attention is not called to this fact. It is to be observed also, that in the case in which the amount of fibrine was normal, the mania supervened on epilepsy, and the mental paroxysms were coincident with convulsive attacks—so that this case is not one of pure insanity.

In all these cases, therefore, as will appear in the subjoined table, there is, with only two exceptions, a material deficiency of fibrine in the blood. Of Dr. Hittorf's seven cases, in six the amount of fibrine stands 1·3, 1·4, 1·8, 1·9, 1·9, 2; and in the seventh, where this substance is in excess, the excess is very trifling. Of Dr. Erlenmeyer's three cases the fibrine stands 1·7, 1·8, in two; and in the third the quantity is normal, being 2·3. While of our own six cases, the fibrine *in all* falls considerably below the normal standard, and in one very considerably so, the numbers being 1·9, 1·7, 1·6, 1·5, 1·5, and 0·06. Surely these results express something more than an accidental coincidence.

Having now offered such materials as can be obtained, it may be well to refer to all the cases of acute and recurrent mania in which the blood has been analysed, and for the more ready view place them together in the following table.

Name and Occupation.	Sex.	Age.	Constitution and Temperament.	Causes.	Present Condition.	Duration.	Previous Treatment and Diet.	Condition of Fibrine and Blood during attack.	Subsequent Treatment.	Condition of Blood on recovery from attack.
Draper's assistant	M.	21	Spare habit, excitable temperament	On father's side, direct hereditary predisposition, no exciting cause; insanity in three generations	Acute mania, irritable, violent, rambling, incoherent	1 month	—	Fibrine, 1·74 Blood-glob. 149·74	Morphia for months without benefit. Hyoscyamus, nourishing diet	Recovered nine months after admission. Fibrine, 2·20 Blood-glob. 110·07
Drum-major in Guards; married	M.	33	Steady, temperate, strong built, muscular, active sanguineous, shy and retiring habits	Not hereditary, intense study at music, ambitious	Acute mania, delusions, noisy, mischievous, and replaced by dementia; self abuse	4 months	Under treatment in military hospital; antiphlogistic.	Fibrine, 1·55 Blood-glob. 125·48	Nourishing diet. Morphia, sedatives	Fibrine, 2·75 Blood-glob. 140·61
E. R.; married, 4 children, youngest 1 month	F.	27	Sanguineous temperament, strong passions, and sexual excitement	Direct hereditary on mother's side, debility and prolonged lactation, puerperal excitement	Acute mania, sullen, inert, violent when opposed, dirty, excited, mischievous	Previous attacks at each nursing, 1 week before admission.	—	Fibrine, 1·67 Blood-glob. 104·68	Tartarized antimony for some weeks with aggravation. Morphia and good diet	Recovery in nine months. Fibrine, 1·75 Blood-glob. 109·32
E. I. G.; single	F	58	Spare habit, shy, retiring disposition	Extreme grief at death of father	Recurrent mania, paroxysms of most revolting character, succeeded by calmness and perfect sanity; regular periodicity of attacks	11 years	—	Fibrine, 1·58 Blood-glob. 121·77	No medicines	Fibrine, 2·88 Blood-glob. 126·01
F. B.; single	F.	47	Sanguineous temperament, great sexual passion	Direct hereditary, mother died insane, leaving 5 children, 4 of whom insane	Recurrent mania, violent paroxysms, quiet and amiable on recovery, no fixed regularity in attacks	20 years	—	Fibrine, 0·06 Blood-glob. 123·81	—	Fibrine, 2·46 Blood-glob. 139·05
W. D., merchant's clerk	M.	46	Phlegmatic, inert	Sorrow or grief at death of father	Recurrent mania; irritable, passionate, and dangerous during attack; bright, intelligent, and rational during recovery	17 years	—	Fibrine, 1·96 Blood-glob. 135·56	—	Fibrine, 2·97 Blood-glob. 121·65

HOOD, BETHLEHEM.

Name and Occupation.	Sex.	Age.	Constitution and Temperament.	Causes.	Present Condition.	Duration.	Previous Treatment and Diet.	Condition of Fibrine and Blood during attack.	Subsequent Treatment.	Condition of Blood on recovery from attack.	
—	M.	66	Strong, robust, plethoric	Loss of money, melancholia	Recurrent mania, violent, had several attacks of epistaxis, intense thirst, diarrhœa, and cough	3 years	—	Fibrine, 1·85 Blood-glob. 114·12	—	—	ERLENMEYER.
Weaver	M.	22	Stout, robust	Maternal grandfather died insane, and other relatives insane; had epilepsy at 14	Recurrent mania, with epileptic paroxysms	8 years	—	Fibrine, 2·30 Blood-glob. 105·59	—	—	
Shoemaker	M.	34	Short, thick, florid	Attacks of epilepsy following blows on head since æt. 16	Recurrent mania, with epilepsy, paroxysms intense	—	—	Fibrine, 1·72 Blood-glob. 118·54	—	—	
Girl	F.	20	—	—	Acute mania	—	—	Fibrine, 2·08 Blood-glob. 109·19	—	—	HITTORF.
Girl	F.	23	—	—	Acute mania	—	—	Fibrine, 1·93 Blood-glob. 116·96	—	—	
Married	F.	37	—	—	Acute mania	—	—	Fibrine, 3·17 Blood-glob. 119·57	—	—	
Unmarried	F.	30	—	—	Acute mania	—	—	Fibrine, 1·92 Blood-glob. 112·01	—	—	
—	M.	30	—	—	Acute mania	—	—	Fibrine, 1·39 Blood-glob. 118·19	—	—	
—	M.	47	—	—	Recurrent mania	—	—	Fibrine, 1·87 Blood-glob. 137·89	—	—	
—	M.	48	—	—	Acute mania	—	—	Fibrine, 1·45 Blood-glob. 130·80	—	—	

CASES

OF

CEREBRAL AFFECTION

CAUSED BY

DISEASE IN THE REGION OF THE NOSE AND EYES.

BY

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It is not of very rare occurrence that we meet with cerebral affections caused by disease of the internal ear; while diseases existing in other regions adjacent to the cavity of the skull are seldom found to excite serious cerebral symptoms.

It is on this account that I venture to communicate the following cases, although I am aware that the observations might be more accurate, and that the manner in which I interpret the phenomena may be contested.

The first case occurred to me while I was assistant-physician to the Clinique at Bonn, then under the direction of the late Dr. F. Nasse, with whom I attended the patient.

Case 1.—*Summary: Erysipelas of face, especially right eye and nose. First appearance of brain-symptoms on ninth day from commencement of illness; death on the twentieth day from same date. Post-mortem. Sero-purulent effusion* *in the lateral ventricles; meningitis on lower surface of anterior lobe of right hemisphere; thrombosis of cavernous sinus and ophthalmic vein of right side.*

F. M—, æt. 25, shoemaker, pale and delicate-looking, had, three years ago, severe rheumatic fever; during the last two years heart-symptoms, with a double bruit over the middle and right half of the sternum, attributed to stenosis and insufficiency of the aortic valves. In February, 1850, he had erysipelas of the face and head, from which he quickly recovered. On December 14th of the same year he was again attacked by erysipelas of the right cheek and right eye. The treatment consisted in the local application of a strong solution of nitrate of silver, and in the administration of effervescent draughts.

At the expiration of a week the erysipelas had almost disappeared, and the patient was considered convalescent, when, on December 23d, he began to complain of a dull headache, the temperature of the whole body being at the same time increased; pulse 105. During the following days the headache became more violent, occasional delirium appeared, grinding of teeth during sleep, twitching of muscles; rather contracted, almost immoveable pupils; sickness and constipation, with a slow and irregular pulse (60—85).

On December 27th patient became drowsy; the pupils wide and sluggish; left arm slightly paralysed.

On December 30th drowsiness much increased; pupils dilated and uninfluenced by light; the paralysis of left side extending now to leg as well as arm. The degree of this paralysis appeared to vary at different times, but a remarkable transitory improvement, as well of this symptom, as of all the others, occurred on December 31st, when the drowsiness almost disappeared, the patient became conscious and was able to speak to his friends; in the following night, however, the coma returned, the pulse became very weak and frequent, the breathing irregular, until death ensued on January 2d, 1851.

The treatment adopted after the commencement of the head-symptoms consisted in local depletion by means of leeches, in the application of ice to the shaved head, of a blister to the neck, and the occasional administration of calomel and jalap.

Post-mortem, twenty-six hours after death.—The upper part of the nose, the lids of the right eye, and the surrounding tissue slightly swollen; a moist, light-brown scab from an almost dried blister is seen on the right side of the nose; a similar one on the upper lid of the right eye. After the removal of the skull, the upper surface of the dura mater appears normal, as also that of the other meninges, with the exception of very slight œdema of the pia mater on both sides. In raising the anterior lobes, the inferior surface of the right hemisphere is found to be covered with a layer of puriform fluid, from the anterior end to the Sylvian fossa. The membranes underneath the puriform fluid are considerably thickened and congested, and cannot be removed without tearing away particles of the subjacent gray substance, which is likewise much congested, of darker colour than in other regions of the brain, and is seen (through the microscope) to contain, besides the normal elements, a great proportion of congested capillaries, many granular corpuscles, and small granules. The puriform fluid exhibits pus-globules of the usual shape. The lateral ventricles are rather dilated, and contain about two ounces of a turbid fluid; their walls are slightly softened. The turbid fluid contains many pus-globules, some large cells like those forming the epithelium of the choroid plexus, and minute granules and fat-globules. The softened substance of the walls exhibits fat-globules of various shapes, granules, and granular corpuscles. The choroid plexuses are much swollen, of dark-red colour; their tissue is denser than usual. The rest of the brain is normal, as also the cerebellum and medulla oblongata. The right cavernous sinus feels hard and enlarged; the walls are thickened; it is filled by a grayish-red coagulum, strongly adherent to the lining membrane, which appears not quite smooth. The coagulum is composed of several layers; the exterior layer being grayish-red, the next being of purer red colour, the centre being the softest, of cherry-red colour, and of the consistency of a stiff paste. The exterior layer is rather crumbly, *i. e.* not elastic; the second exhibits more elasticity and cohesiveness. The internal carotid and the nerves within the walls of the sinus appear in their tissue unchanged. The portion of the ophthalmic vein in immediate connexion with the sinus contains a dark coagulum, rather firmly adherent to the thickened lining membrane. The blood in the circular sinus is coagulated; that in the left cavernous sinus is not quite coagulated; the walls are of normal appearance. The other sinuses of the dura mater exhibit nothing pathological. The left ventricle of the heart is hypertrophied. The mitral valves are normal; the semi-lunar valves of the aorta are thickened, rather rigid, and contracted. On the pericardial surface of the heart are several white, tendinous patches, of large size. The lungs and the organs of the abdominal cavity are normal; there are nowhere any secondary abscesses.

The brain-symptoms in this case resemble in many respects those caused by acute hydrocephalus, which had, in fact, been diagnosed by two medical men, who had examined the patient, without being acquainted with the history of the case. The changing character of the hemiplegia is remarkable, and not less so the striking improvement of all the symptoms two days before death; but this latter phenomenon is occasionally seen to occur, in the most deceptive manner, in other diseases of the brain, although the organic lesions remain unaltered.

The fact that erysipelas of the face, and especially of the eye and nose, preceded the appearance of the head-symptoms, coupled with the pathological condition of the right cavernous sinus and ophthalmic vein, as also the existence of purulent meningitis on the inferior surface of the right anterior lobe, scarcely admits of a doubt that the endocranial affection had been caused by the erysipelas of the face, and that the ophthalmic vein had been the medium of communication. There may be, however, some difference of opinion regarding the exact way in which this communication was effected. I should be inclined to think that, through the ophthalmic vein, some blood, contaminated with erysipelatous matter, was carried to the cavernous sinus, where, favoured by the peculiarities of the sinus, it led to coagulation, which was propagated to the contents of the ophthalmic vein. The irritation caused by the coagulum, and the changes occurring in it, led to inflammation of the walls of the sinus and vein (phlebitis), which inflammation may have extended itself to the vessels of the meninges of the lower surface of the anterior lobe and to those of the choroid plexus.

This is, however, not the only way in which the endocranial alterations may have been effected. Thus it may be supposed that the coagulation of the blood commenced in the capillaries and venous plexus of the eye and nose, and proceeded thence to the ophthalmic vein and cavernous sinus.

The inflammatory thickening of the coats of the cavernous sinus and ophthalmic vein might also lead to the view that phlebitis had been the first link in the chain of the pathological processes, and that the coagulation and further alteration of the blood within the sinus were only the consequences of the inflammation of its walls. As we know, however, that the presence of a coagulum in a vein, and the changes constantly taking place within such a coagulum, are in themselves sufficient to cause inflammation of the coats, the existence of the remains of inflammation in the coats of the sinus cannot be regarded as stringent evidence that the erysipelatous process was propagated from the face to the brain, by phlebitis.

The fact that we were not permitted to examine the interior of the eye and nose, prevents me from stating in what condition the veins and capillaries in these regions were.

Case 2.—*Summary: Syphilitic ozæna. Appearance of head-symptoms soon after cessation of sanious discharge; rigors four days later; pyæmia; death on thirteenth day after first rigors. Post-mortem. Thrombosis of cavernous sinus and ophthalmic vein of left side, as also of longitudinal, and, to a lesser degree, of circular and right cavernous sinus. Purulent meningitis on inferior surface of left anterior lobe. Secondary abscesses in lungs and liver; purulent effusion in left pleural cavity.*

F. B—, æt. 24, a waiter, belonging to a scrofulous family; had, himself, glandular swellings in childhood; was affected with chancre in 1845, when, he states, he was salivated. In 1846 he had ulcers in the throat, for which he again was treated with mercury internally and nitrate of silver locally. In 1848 he had psoriasis, which disappeared under the use of "Zittmann's treatment."[1] In 1849 he suffered from "pain and swelling in the bones of the legs," was treated in an hospital with iodide of potassium, and left much improved. Towards the end of the same year, after he had come to England, he had, according to his account, "cold in the head, which became chronic, and led, by degrees, to a considerable discharge of yellowish matter of a disagreeable odour." At times, he said, the secretion was scanty, and then he felt as if he was "stuffed up," *i. e.* he had a sensation of dulness, heaviness, and pain in the forehead, and could not breathe through the nose. Since March, 1851, the nose has become changed in shape, viz., sunk in the region of the bridge. Once or twice, small pieces of bone were discharged with the matter, which was occasionally mixed with blood. Of late he had been under homœopathic treatment.

On November 28th, 1851, F. B— came as out-patient to the German Hospital. The patient had a pale, sickly appearance; the nose was considerably depressed and flattened in the region of the bridge; there was a marked fetor from both nostrils, and much discharge of sanious fluid. Pressure on the nose not painful; appetite in general good; sleep sometimes sound, sometimes disturbed by headache. Ordered iodide of potassium internally, and a weak solution of chloride of zinc for injection.

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[1] Zittmann's treatment consists principally in the administration of a decoction of sarsaparilla and senna. Low diet forms part of this method of treatment.

December 12th.—The discharge is diminished, and less fetid; the appetite improved; general aspect more healthy. Treatment as before.

After this date the patient did not come back to the hospital; feeling much better, he thought he could do without the further use of medicine. The discharge, however, had never entirely ceased, and the fetor, too, had not altogether disappeared.

When I was requested to see him on February 4th, 1852, he stated, that about ten days ago he had been exposed to cold; that soon afterwards, on January 25th, he suffered from a dull headache, and observed, at the same time, that the discharge from the nostrils was much diminished.

On the 29th he was seized, during the night, with rigors, followed by heat and violent headache.

On the 30th he felt much better, and slept also well during the following night.

On the 31st he was at work; but during the succeeding night the rigors returned with increased force, and were accompanied and followed by almost intolerable headache, spreading from the forehead over the whole head. The pain abated towards daybreak, but did not altogether disappear during the day. No rigors returned in the night from February 1st to 2d, but the patient was not free from headache. After 10 p.m. on February 2d, he was again attacked by rigors and violent pain in the head. He felt more easy on February 3d, and supposing his illness to be ague, with which he had been affected some years before, he treated himself with quinine.

Condition on February 4th, in the evening: Patient's principal complaint is headache, especially in the frontal region, rather increased by shaking the head, not increased by tapping on the forehead. The head is hot, but its temperature is scarcely augmented over that of the rest of the body. Nose flattened and slightly swollen; nostrils blocked up by dry, brownish crusts; fetor moderate, but quite distinct; pressure on nose not painful. Both pupils of equal diameter, contracting well under the influence of light. Bowels costive. Pulse regular, about 90.

Treatment.—Removal of crusts. Leeches to nostrils. Tepid injections. Mistura Sennæ cum Magnesiæ Sulphas.

7th.—Appearance of nose unchanged. Headache constant, but during night increased. Patient was yesterday often delirious; to-day he is drowsy, but can be roused, and then answers reasonably. Pupils since this morning rather dilated and sluggish. Had repeated rigors. Spleen was yesterday found perceptibly enlarged; to-day there is, in addition, tenderness of the lower part of the left side, and a soft friction sound is occasionally heard.

9th.—Somnolency increased; carphology; muttering delirium; frequent contractions of single fascicles of muscles, especially those of the thighs; ptosis of left upper eyelid; left eye moves only from inner corner to centre, never to outer corner of the orbit; both pupils wide and sluggish. Tongue dry, friction sound over lower part of left side again noticed; dulness increased. Pulse 120 to 125, very small.

10th.—Complete coma; right-sided hemiplegia. Left eye is almost closed, the conjunctiva slightly injected.

Death on February 11th.

Post-mortem, twenty-two hours after death.—The examination of the interior of the eye and nose was not permitted. Upper surface of both hemispheres normal; the inferior surface of the left anterior lobe is covered with a thin layer of pus, which is especially distinct along the sides of the veins. The reaction of the pus is alkaline. The meninges over both hemispheres are congested; but underneath the layer of pus on the left side much more so than in other regions; they are, at the same time, thickened; the large veins appear distended, and are filled with dark, coagulated blood; these thickened membranes cannot be removed from the subjacent brain without taking away part of the gray substance, which, here, is softer and of deeper colour than elsewhere. This altered gray substance exhibits under the microscope many granular corpuscles, single granules, fat-globules, injected capillaries, and free blood-globules. The lateral ventricles of the brain contain about three drachms of not quite transparent fluid; their cavities are not enlarged, the walls not softened, the choroid plexus not congested. The other parts of the brain, the cerebellum and medulla oblongata, offer nothing abnormal.

The superior longitudinal sinus contains a dark-red coagulum, rather firmly adherent to the walls, which, however, are not perceptibly altered. The left cavernous sinus is filled by a crumbling, slightly cohesive substance of reddish-gray colour. The external part of this thrombus is closely adherent to the walls, the centre is formed by a pultaceous fluid, of the consistence of thick pus, and of brownish-red colour. The walls of the sinus are considerably thickened, as are also those of the central end of the ophthalmic vein attached to the sinus, which is likewise filled by a dark, rather dry coagulum. The internal carotid artery and the nerves on either side of the walls of the left cavernous sinus appear not materially changed, with the exception of increased thickness of the surrounding areolar tissue. The blood in the left inferior petrosal sinus is coagulated, of dark colour, but can be easily separated from the walls, which are not thicker than usual. The circular and the right cavernous sinuses contain likewise a dark-red coagulum, which is but slightly adherent to the unchanged lining membrane. The other sinuses of the dura mater contain a loose coagulum, which is not at all adherent to the walls.

The bone forming the sella turcica and surrounding region of the skull presents no appearance of carious destruction; the dura mater is nowhere separated from it, there is nowhere a trace of pus between the bone and dura mater. The fluid in the centre of the thrombus of the left cavernous sinus contains—1st, small granules, which are dissolved by acetic acid; 2d, aggregations of such granules into irregular-shaped bodies (round, oval, crescentic), which are likewise dissolved by acetic acid, nowhere exhibiting the characters of pus-globules; 3d, other granules and aggregations of granules, dissolvable by ether and not by acetic acid; 4th, oil-globules of various size; 5th, blood-globules of different shape—some being much shrunk, some exhibiting a tendency to the star form, others being granular on their surface.

The left pleural cavity contains about a pint of thin, opalescent fluid, of a reddish hue and slightly acid reaction; flakes of plastic substance are swimming in this fluid, and others are loosely attached to the pleura.

There are several spots of secondary inflammation in both lungs.

Heart flaccid, otherwise normal. The liver exhibits three spots of the size of a walnut and smaller, hard in the circumference, with commencing suppuration in the centre. Spleen enlarged, of pulpy consistence. The other organs of the abdomen are normal.

Although these two cases differ considerably, as well with regard to the original complaint, as also to the symptoms of the cerebral affection and the post-mortem appearances, yet they seem to be closely related to one another in the manner of propagation of the disease from the exo- to the endo-cranial regions. As I was in neither case allowed to examine the interior of the nose and eye, I am unable to state the exact seat and extent of the original disease. The appearance of the nose and the history of the disease in the second case show that there had existed for a considerable time necrosis of several bones of the nose. The vomer and the nasal bones were evidently to a great extent destroyed; the profuse secretion renders it probable that the disease had spread considerably in the adjacent regions.

The endocranial affection appears in this instance, too, to have commenced with coagulation of blood (thrombosis) of the cavernous sinus, in consequence of the introduction of morbid substance from the original seat of the disease, through the ophthalmic vein and its capillaries. The coagulation of the blood in the ophthalmic vein itself seemed to us of later date, as the changes in the coagulum were less advanced. The thickening of the walls (phlebitis) I am here again inclined to interpret as the consequence of the thrombosis and the changes taking place within the thrombus. The meningitis, too, and the congestion of the veins on the inferior surface of the left anterior lobe, appear to me caused by the thrombosis of the sinus, into which the veins of this region carry their blood. The obstruction of this passage might by itself lead to congestion and inflammation; it is, however, not to be overlooked that, besides this mere mechanical element, the quality of the decomposition occurring within the contents of the sinus, may have also contributed to the coagulation of the blood in the veins, and to the inflammatory processes within the pia mater and subjacent part of the brain.

The microscopic appearance of the thrombus, as well in this as in the previous case, exhibits no proof of the introduction of actual pus-globules into the blood from the seat of the original disease, but only the results of the changes usually going on within a thrombus. The view that what we term "pyæmia" is always caused by the entrance of entire pus, viz., serum and globules, into the circulation, is so untenable, that it is unnecessary to dwell on this subject.

With regard to the diagnosis, the consideration of the history of the patient, of the headache, the rigors, and the whole aspect of the case, could not fail to point towards the real nature of the case. The intermittent character of the pain, the periodic occurrence of the rigors, were in the present instance very striking, but they are often met with in similar affections, especially as the consequences of internal otitis. The dropping of the upper lid of the left eye, as well as the paralysis of the left external rectus, are easily interpreted by considering the relation of the third and sixth nerve to the cavernous sinus.

The two following cases differ considerably from those preceding, but bear great similarity to each other :

Case 3.—*Summary : An infant is affected with purulent discharge from left nostril and left eye from birth. At the age of ten weeks cessation of the discharge, followed within a few days by epileptiform fits and coma, which disappear soon after the return of the discharge.*

A. K—, a male child, well developed, had, very soon after it was born, what was considered a cold, showing itself in a discharge of yellowish fluid from the nose, and in an inflammation of the left eye, which was almost always closed and discharged likewise a purulent fluid. When I saw the child first, on March 21st, 1859, it was three weeks old; it then was well nourished ; the left eye was red and swollen, and entirely closed ; there was much purulent discharge from between the lids. The nose was swollen and stuffed up with crusts and muco-purulent fluid, the left nostril completely, the right less so ; the child breathed through the mouth. There had always been much discharge from the nose, sometimes watery, sometimes thick and yellowish, sometimes mixed with blood ; this discharge came almost entirely from the left nostril. General health good. Pulse 115 to 118. On further inquiry, it was found that the mother had been affected, during the last weeks before her confinement, with a rather profuse discharge of yellowish matter from the vagina, and that the child, on account of the nurse's late arrival, had not been washed until three hours after it was born. To these circumstances I ascribed the inflammatory affection of the left eye, and of the interior of the left nostril.

Treatment.—A weak solution of sulphate of zinc to be frequently applied to the eye, and dropped into the nostril, by means of a sponge.

April 14th.—Eye more open, lids less swollen. Discharge from nose varies. On the nates are several red pimples, and some round, slightly elevated patches of bright-red colour. On this day I had the advantage of seeing the child and mother together with Dr. Oldham, who found excoriations on the os uteri of the mother, and relaxation of the anterior wall of the vagina. Dr. Oldham was likewise of opinion that the affection of the child was due to the discharge from the mother. Although we had no proof of the specific nature of the affection, we prescribed the fourth part of a grain of Hydrarg. c. Creta to be given twice a day. The child was, further, advised to be weaned.

May 1st.—The spots on the nates have disappeared, but there is no change in the condition of the nose. Bowels frequently moved, motions greenish, containing much mucus.

Treatment.—Omit Hydrarg. c. Creta. Tepid water to be frequently squeezed into the left nostril.

The discharge from the nose is reported to have been much diminished on May 6th, and to have altogether ceased on the 8th. At 9 a.m. of this day, when the mother considered her baby to be in improved health, the nurse observed, while bathing the child, that the right leg was drawn over the left, and the right arm over the chest; this was rapidly followed by stiffness of the whole body, foaming from the mouth, and clonic convulsions. These fits returned six times within four hours. At 1 p.m., when I saw the child together with Mr. R. Fish, with whom I had the pleasure of attending the case, we found it asleep; the sleep was, however, uneasy, the child starting often; breathing irregular, rather accelerated; pulse 140; skin moderately warm; pupils slightly dilated, sluggish; bowels had been moved twice in the morning. When awake the child seemed to take no notice.

At 8 p.m. of the same day the fits had become very frequent, the convulsive movements scarcely ever ceased. One of the fits which I witnessed commenced with sudden stiffness of the whole body, especially of the right arm and leg; the thumb and fingers of the right hand being so forcibly bent in, as to leave the marks of the nails; the eyes at first turned upwards and then rolling about, the pupils being wide and inactive; the face livid; the pulse at wrist not perceptible; the general stiffness being soon followed by clonic convulsions, more strongly marked on the right side; mouth covered with foam. The convulsive movements cease after about ninety seconds with a deep sigh. The face and head are then seen covered with perspiration, and a restless sleep succeeds.

Treatment.—Half a grain of calomel every eight hours; small doses of ether and sal volatile every three or four hours.

May 9th, 9 a.m.—Many fits during night. The convulsive movements scarcely ever cease; still more marked on the right side. Pupils wide, scarcely influenced by candle-light; pulse 160, very weak; breathing irregular; bowels moved repeatedly. No food of any kind has been taken since yesterday.

Treatment.—Wet nurse. Hot bath. Calomel to be omitted. Ether and sal volatile to be continued.

9 p.m.—Soon after this morning's report, while in the hot bath, the child sneezed several times, by which much discharge was expelled from the nose. No fits since then. Child sleeps much; has taken the breast. Pupils less wide. Pulse only 125.

10th, 2 p.m.—Many fits since this morning; scarcely any discharge from nose. Pupils sluggish. Pulse 135. Omit calomel.

9 p.m.—Convulsions almost without interruption until a few minutes ago, when after a hot bath the discharge had again become copious, and the child fell asleep.

11th.—No return of fits. Much sleep, with frequent starting; child starts also, when awake, at every noise. Convulsive movements are frequently observed in right arm. Pupils contract well. Pulse 120. Much discharge from nose.

13th.—Yesterday evening, and during last night, several fits. Secretion from nose scanty. Injection of warm water into left nostril; frequent application of moist sponge to nose.

14th.—Discharge much more copious. No fits except two, which seemed brought on by the shock of the injections.

During the week following the last report, the child had still several fits, which, however, appeared to be excited by sudden noise. The treatment consisted in small doses (one fourth to half a grain) of oxide of zinc and the application of the moist sponge.

After May 21st no fits returned; the child gained steadily in health and strength; the discharge from the nose became, by degrees, much diminished, but did not altogether cease until ten months later. Besides the liberal use of warm water by means of the sponge, nothing was done.

Case 4.—*Summary: A child is affected with congenital syphilis and ozæna; cessation of the sanious discharge from the nose is followed by epileptiform fits, coma, and other cerebral symptoms, which rapidly disappear after the re-establishment of the discharge.*

M. G— came first under my observation in October, 1858, as an out-patient at the German Hospital. The little boy was five months old, rather emaciated, of sallow complexion, suffered from chronic diarrhœa, and had large mucous patches, of yellowish colour, near the anus, and several sores of the size of a threepenny-piece, with livid margin, on the nates. The father of the child had had chancre ten years ago, and later secondary syphilis. The mother has had several times, after her marriage, soreness of the lips and mouth; lost the only other child from chronic hydrocephalus, with symptoms of congenital syphilis; miscarried once.

The treatment of the child consisted in small doses of Hydrarg. c. Creta and opium, and after the cessation of the diarrhœa in Hydrarg. c. Creta alone. The mucous patches and sores disappeared within five weeks; the child was, at the same time, in every other respect so much improved, that the mercury was left off, and Oleum Morrhuæ substituted. After March, 1859, the mother did not bring the child any more.

Since the end of June, 1859, the child is said to have always had a "cold in the head;" sometimes the nose was "stuffed up" for days; at other times there was considerable discharge of "fetid matter." The child is reported to have been more cheerful when there was much discharge, irritable and unhappy when there was none.

On October 17th the patient was seen again. He was not emaciated, but of sallow complexion; the nose was swollen; there was from both nostrils, especially from the right, discharge of a sanious, yellowish fluid, with much fetor. Pressure on the bridge of the nose seemed to cause pain.

Treatment.—Good nourishment. Three times a day two grains of iodide of potassium in a mixture. Frequent use of tepid fomentations and injections.

November 4th.—The child breathes more easily through the nose; the secretion is less fetid, and diminished in quantity. General health improved.

After this date the child was not seen again until December 17th. The mother considered the improvement so great, that she soon discontinued the mixture. In the beginning of December, however, the discharge became again increased in quantity, and more fetid. On December 15th, the little boy began to cough, became restless and feverish, breathed always through the open mouth, and took no food.

On December 17th, in the morning, the child had an attack of convulsions, which lasted several minutes. The fits recurred several times in the course of the day. At the time of my visit, at 5 p.m., an hour after the last fit, I found him lying on the back, sleeping, with mouth wide open, and deep, snoring respiration. The nose is almost closed by brown crusts, there is no discharge from it; the mother does not know when it ceased, but is sure that it had not done so three days ago. The right eye seems smaller, the lids being slightly swollen. Pupils rather wide, but contracting under the influence of light. Head warm, but not more so than rest of body. Right arm moved about in various directions, apparently without influence of will; left arm lies motionless at side of body, but is not quite flaccid, and when placed in an uneasy position, as over the head, it is gradually drawn back. Sensation not impaired. The child is not quite unconscious.

During my visit, while the mother took the patient on her lap, an epileptiform paroxysm occurred, lasting for more than five minutes.

Treatment.—To soften the crusts; to inject warm water into the nose; to apply a leech to the interior of each nostril. To place the head high. Hot bath.

December 18th, 9 a.m.—Before the crusts were removed and the leeches applied, the child had five more paroxysms, and became quite comatose. At 4 a.m. the leeches sucked well. Fits since then less frequent, but coma continues; pupils wide and sluggish; head rather hot; a small quantity of sanious discharge is observed at the nostrils. Movement of limbs as yesterday. Pulse 120 to 125, regular. No motion of bowels within the last forty-eight hours.

Treatment. — Another application of leeches. Two grains of calomel to be given immediately. Continue injections.

18th, 4 p.m.—Injections have not been made for fear of disturbing the child.

Two fits since this morning; comatose state continues. Pulse 125 to 130.

During the injection of tepid water into the nose, which I made myself, a fit occurred, as soon as I commenced the operation. By repeated injections, resumed as soon as the convulsions ceased, a large quantity of thick, fetid fluid, mixed with blood and almost solid pieces of yellowish colour (crusts), was removed, more from right than from left nostril, but much from either.

19th, 9 a.m.—Only two fits since yesterday, the one brought on by the injection, the other by banging of the door. Between the fits the child is conscious; sleeps much; moves limbs of both sides equally. Pupils normal. Pulse 110. Bowels moved several times. Much discharge of purulent matter mixed with blood.

Treatment.—Beef-tea and milk. Warm sponge to be frequently applied to nose.

After the last report no return of epileptiform fits. Under the continued use of iodide of potassium, and of the injections of warm water, and the moist sponge, the discharge from the nose became much diminished, and had almost disappeared in the beginning of February; the general health being at the same time much improved.

The nature of the original complaint was not quite the same in the last two cases; but in both there was chronic catarrh or inflammation of the Schneiderian membrane, much more advanced in one side of the nose than in the other; and the phenomena of the cerebral affection, too, resembled each other much in both.

It seems, further, quite certain that the retention of the discharge—through congestion of part of the membrane, or blocking up of the natural passage by means of dried crusts, or both circumstances combined—was the cause of the cerebral disturbance, as the re-establishment of it was followed almost immediately by the cessation of the principal symptoms. It is true that some fits did occur in both cases after the free passage for the discharge had been restored, but we know that convulsive fits, after having once occurred, often become habitual, especially in children, although the primitive cause has been removed. The last three fits, in Case 4, were evidently caused by external influences and mental agitation, acting on nervous centres inclined to fits; and in Case 3, many fits were occasioned by noise or injections, the nervous centres having previously acquired such a convulsive disposition.

The inference that the retention of the discharge caused the cerebral affection, is strengthened by the fact that, in Case 3, a relapse took place when the discharge from the nose became again, for some time, scanty.

In Case 3 the convulsions were more marked in the limbs of the side opposite to that where the original disease had made the greatest progress; in Case 4 the limbs of the left side seemed incompletely paralysed, while the principal discharge took place from the right nostril. In both cases, therefore, it is permitted to infer that the hemisphere of the brain corresponding to the side most diseased was most affected.

Of what nature the affection of the brain was, which led to the epileptiform convulsions, seems more difficult to understand. It may be conceived that there was, in these cases, as in the first and second, absorption of part of the sanious or purulent matter, through branches of the ophthalmic vein, that this led to stagnation of blood in the cavernous sinus and further disturbance of the circulation of the brain, which in infants, more disposed to convulsive disorders, caused the fits and the comatose state connected with them. The timely restoration of the normal passage for the discharge, may be considered as having prevented the further absorption of morbid matter, and thus having given the opportunity for the recovery of the equilibrium in the endocranial circulation.

The view that real inflammation existed in the brain or meninges of the children, I am not inclined to adopt, as the rapid disappearance of the brain-symptoms appears to me in discord with it.

I feel, however, that other interpretations may be brought forward different from the one I have ventured to give; but I will not further occupy the time of the Society with hypothetical views.

With regard to the diagnosis, prognosis, and treatment of such cases, I shall add only a few words.

A due consideration of the history and the present symptoms of each case will, I should think, in most instances allow us to recognise the connection between the endocranial and the exocranial affection. Although, I confess, in the third case this connection had escaped me, until nature had given a broad hint by the sneezing of the child and its consequences. The more accurate diagnosis, however, of the cerebral, or rather endocranial affection, will, in general, be very difficult. There are as yet, for instance, to my knowledge, no certain symptoms observed as due only to thrombosis of the sinus of the dura mater. Phenomena like the ptosis of the upper eyelid and the paralysis of the external rectus in the second case, are of great importance, as they are occasioned by the affection of the sinus (thrombosis and inflammation of its walls) influencing the third and sixth cerebral nerves in their course through the sinus. These symptoms, however, need not be present, especially in the first days of the thrombosis, as the nerves are protected by their sheaths, and their functions may remain, as we have repeatedly seen, undisturbed for a considerable time, in spite of their being surrounded by pus and other morbid substances. The functions of the third and sixth nerves may further be disturbed by other pathological conditions in, and adjacent to the brain. Yet the symptoms in question are very valuable, and may occasionally, with due consideration of the concomitant circumstances, lead to the diagnosis of affections of the cavernous sinus.

The prognosis must, of course, widely differ according to the individuality of every case. Thus, in Case 1, the symptoms of meningeal inflammation and of inflammatory effusion into the ventricles left little hope. In Case 2 the occurrence of the repeated rigors plainly indicated the existence of pyæmia in its worst degree, and the issue could scarcely appear doubtful. Cases 3 and 4, on the contrary, show that even an apparently severe character of epileptiform paroxysms with continued coma, especially in children, need not be looked on as a fatal sign, as long as the symptoms of other complications, such as meningitis or effusion into the ventricles, have not yet appeared.

Concerning the treatment, we can do little, I fear, in cases of erysipelatous ventricular effusion or general pyæmia. But in cases such as 3 and 4, where the cerebral symptoms have been preceded by a chronic discharge from the nostrils —whether caused by mere chronic inflammation of the mucous membrane, or whether connected with necrosis or caries—the removal of the crusts, the application of leeches to the nostrils, warm fomentations and injections, will be of great service. Internal remedies are of only secondary importance. On the proper use of the syringe I would place the greatest reliance; but it ought, in general, not to be entrusted to the unprofessional attendants, who will very rarely use it with sufficient energy, but the medical man himself should perform the injection. The regular and adequate use of the syringe offers also the best prophylactic means in the various conditions of ozæna; in some of which, of course, the constitutional treatment cannot be superseded by it.

ON

CONGESTION OF THE HEART,

AND ITS

LOCAL CONSEQUENCES.

BY

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The principal object of this paper is to call the attention of the Fellows of the Society to the occurrence of congestion of the muscular tissue of the heart; to the most common and direct consequences of that congestion, viz., induration, toughening, and thickening of the walls of the heart; and to the influence which those changes of texture exercise (as predisposing causes) on the development of permanent dilatation of the heart.

The expression, congestion of the heart, has been hitherto used to signify extreme distension of the cavities of the heart with blood; few pathologists have even mentioned congestion of the substance of the heart, and none have, I believe, so much as adverted to its most important consequences.[1] When the right cavities of the heart are over-distended with blood from an impediment existing to its onward flow, there is not only engorgement with blood of the venæ cavæ and of the veins opening into them, but also of the coronary sinus, veins, and branches. Now, it is a pathological law, that mechanically induced congestion, if long continued, slowly formed, and intermitting altogether or in degree, has for effect induration, and also (when the fibrin exuded is not of the powerfully contractile variety) permanent increase in bulk of the organ or tissue.

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[1] Bertin notices the dilatation of the veins of the heart which accompanies engorgement with blood of the right auricle: "Il (le sang) s'accumule donc dans leur cavité, distend leur parois, et les engorge jusqu'à leurs dernières extrémités." He observes also that rupture of the distended veins may occur. ('Traité des Mal. du Cœur,' 1824, p. 41.)

Thus, when the inferior vena cava is unable to pour its blood freely into the right auricle, the liver and kidneys are at first enlarged and softened from over-distension of their vessels and effusion of serosity into their structures. If the impediment to the onward flow of the blood be quickly removed, those organs soon recover their normal size and consistence; but, if the impediment continues for any length of time, then the organs in question are found to be indurated, toughened, and (except under the conditions previously specified) permanently enlarged. The induration, toughness, and enlargement, are due, chiefly at least, to an interstitial exudation of lymph, which is found after death, either amorphous, or in the form of granules, or more or less organized into fibrous tissue. It may be that, in particular cases, there is also some true hypertrophy of the proper structure of the congested part.

When the contractile coats of a hollow viscus are indurated, toughened, and thickened by an interstitial exudation of lymph, their normal contractile power is diminished. The consequence is, that although the resistance to over-

Rokitansky has the following remarks on the subject at page 267 of the second volume of the last edition of his 'Path. Anat. § 7: Krankheiten der Textur.—Hyperämie:' "Ein Strotzen der Gefässe des Herzens, zumal seiner Venen und kleine Blutaustretungen in Form von Hirsekorn—Linsengrossen Ecchymosen unter dem Pericardium in der Nähe des Sulcus transversalis, an den Verhöfen und an den Ursprungstücken der Arterienstämme kommen bei Stenosen des Herzens und bei Asphyxien Neugeborner und Erwachsener häufig vor."

distension of the walls of the viscus is greater than natural, yet, if the distending force be sufficient to overcome that resistance, and be repeatedly applied at short intervals over a length of time, permanent and extreme dilatation of the viscus will be produced.

What is true in these respects of other organs and tissues is true of the walls and cavities of the heart. When there is long-continued obstruction to the passage of the blood out of the right ventricle, and consequently impediment to the free entrance of the blood from the coronary sinus into the right auricle, congestion of the walls of the heart follows. If the impediment to the exit of the blood from the sinus be slowly produced, be moderate in degree and permanent, or be frequently repeated, then induration, toughening, and thickening of the walls of the heart will ensue, and permanent dilatation of its cavities be the final result; the over-distension of the walls of the cavities being the immediate cause of the dilatation—the induration, toughening, and thickening being the cause of the permanence of the dilatation.

The peculiarities of the indurated and toughened walls of the heart are as follows:

When divided across, the outer wall does not fall inwards, the form of the cavity being still retained, even though, as is sometimes the case, the walls are (in consequence of the extreme dilatation of the cavity) thinner than natural; the cut surface of the muscular tissue has a very smooth, compact, homogeneous appearance; the columnæ carneæ stand firmly out; the tissue is harder than natural, and singularly tough. This tough, leather-like quality is one of the most marked characters. The colour of the indurated, toughened tissue may be paler or darker than natural.[1]

The microscopical appearances of the indurated and toughened tissue are—

1st. The striæ of the muscular fibres are generally indistinctly seen, though here and there they are as perfect as in health.

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[1] Laennec described several of these characters as proper to the dilated and hypertrophied heart. De l'Auscult. Méd., vol. ii, p. 514.

2d. The muscular fibres are more firmly united to each other than in the healthy heart.

3d. Both between and within the muscular fibres are innumerable molecular granules, chiefly protein.

4th. Lymph which has a granular form exhibits in the heart, as elsewhere, a tendency to undergo fatty degeneration, and the muscular fibres of the heart, damaged by the congestion and the presence in and among them of the lymph, are prone to the same change; the consequence is, that it is common to find here and there a considerable number of particles of free olein as well as muscular fibres which have undergone fatty degeneration.[1]

5th. In some cases the cellular or connective tissue seems to be increased in quantity.

When the exit of the blood from the right auricle is suddenly or greatly impeded, it is not unusual to find small crimson spots studding the external surface of the heart, and less commonly the internal surface. These spots are evidently extravasations of blood, the result of capillary hæmorrhage from mechanical obstruction to the onward flow of the blood.[2] Under like conditions there is often found a considerable excess of serosity in the pericardium, and œdema of the loose cellular tissue at the base of the ventricles; the latter especially, if the patient is much emaciated.

When the congestion has been extreme and long continued, the coronary sinus is found to be more capacious than natural.

In studying the effects of congestion on tissues and organs, it is necessary to separate not only slight from extreme congestion, but also continuous from intermitting congestion, suddenly formed from gradually formed congestion, and congestion of organs the action of which is normal or increased from congestion of organs the functions of which are less actively performed than in health.

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[1] When the fatty metamorphosis of the muscular tissue and lymph is extensive and extreme, softening of the walls of the heart may ultimately occur; the parietes of the heart being first indurated, and subsequently softened, in consequence of the indurated tissue undergoing fatty degeneration.

See Rokitansky, in previous note.

It is the very gradually developed, long-continued, intermitting congestion of organs the functions of which are over-actively performed notwithstanding their congestion, which is especially followed by induration, toughness, and hypertrophy.

The causes which lead mechanically to an accumulation of blood in the right cavities of the heart have been attentively studied, and are well known. They are referable either to disease of the arteries of the heart itself, or to some cause interfering with the free passage of the blood through the vessels of the lungs.

The following cases are sufficient to *illustrate* the main facts adverted to in the foregoing remarks; viz., the most common causes of mechanical impediment to the passage of the blood through the right side of the heart; the relation between those impediments, engorgement of the right cavities of the heart, and congestion of the walls of the heart; the result of that congestion of the cardiac parietes, viz., induration and toughness, especially affecting the parts most in action; and the influence of induration and toughening of the walls of the heart in the production of permanent dilatation of its cavities.

Case.—A woman, æt. 50, suffered during life from general anasarca, symptoms of extreme congestion of the lungs, liver, kidneys, &c. After death, the walls of the heart were found greatly thickened, and the cavities dilated. When the heart was opened the divided walls still retained their rounded form; they did not fall inwards; the substance of the organ was remarkably tough and hard; the mitral orifice was much constricted; the left auricle was scarcely larger than natural, the right was very capacious; the foramen ovale was so patent as to admit the points of three fingers; the right auriculo-ventricular orifice was very large (five inches and a quarter in circumference); the coronary sinus and veins were considerably dilated. The orifice of the aorta seemed of normal size, it measured two inches and five eighths in circumference; the orifice of the pulmonary artery was so much dilated as to measure four inches and a half in circumference. There was a large amount of subendocardial and subpericardial extravasation of blood in very thin layers, varying in extent, at some places forming mere specks, at others large patches.

The kidneys were hard, tough, and granular. The liver was remarkably tough and granular, and fibrous at the margin.

The lungs were the seat of pulmonary apoplexy.

When examined by the aid of the microscope, every fibre of the heart was found to be studded, both within and without, with minute granules—the majority protein, a few olein. The fibrous tissue seemed to be more abundant than natural. The cross-markings of the fibres were less distinct than are those of the fibres of a healthy heart.

Remarks.—The primary disease in this woman was evidently narrowing of the left auriculo-ventricular orifice. The blood, thus prevented from readily passing into the left ventricle, must have exerted abnormal pressure on the septum auricularum; as a consequence, it is probable that, an obliquely patent foramen ovale, such as is so often found when no admixture of the blood occurs, became dilated. The pressure on the two sides of the septum being unequal, the blood must have passed from left to right auricle, thus accounting for the difference in the relative size of the two auricles. The large size of the pulmonary artery was, doubtless, due to the impediment to the escape of blood from the pulmonary veins, and to its passage through the lungs.

These changes, as well as the condition of the liver and kidneys, point to the long continuance of the impediment to the circulation; the large size of the veins of the heart, and the extravasation of blood under the visceral pericardium and under the endocardium, show the degree to which the substance of the heart must have been congested during life; the toughness and hardness of the tissue of the walls of the heart were singularly well marked.

Case.—J. S—, æt. 35, a man of intemperate habits, by trade a carpenter, was admitted into University College Hospital, November 14th, 1860.

On September 6th he had an epileptic fit. On the 16th he was seen by Dr. Coghlan, of Notting Hill, who found, in addition to much cerebral disturbance, a loud, soft, blowing, systolic cardiac murmur, having its point of greatest intensity at the apex; the heart's impulse abrupt and irregular in force and frequency; the pulse at the wrist so rapid, weak, and irregular, that it could not be counted. No anasarca.

Against the advice of his medical attendant the man resumed work, and when again seen he was suffering from extreme dyspnœa and extensive dropsy.

On admission into the hospital, about six weeks after the supposed commencement of his illness, the most prominent symptoms were extreme dyspnœa, anasarca, and ascites.

All the superficial veins were distended with blood. The jugular and subclavian veins became distended to the utmost when the man coughed; they pulsated synchronously with the heart's beat; a strong thrill was perceptible by the finger placed over the point of junction of the right internal jugular and subclavian veins. On slight pressure both the pulsation and the thrill ceased.

Physical signs indicated the existence of hypertrophy and dilatation of the heart, and regurgitation of blood through the right and left auriculo-ventricular orifices. The urine contained a considerable quantity of albumen. The congestion of the various organs and tissues diminished very greatly during the first few weeks of the man's stay in the hospital; so much so that the anasarca almost disappeared; there was scarcely a trace of albumen in the urine; the pulse fell to 96, and was more regular in force and frequency, and the man could lie on his back, and walk about the ward without difficulty.

For a fortnight before the man's death, on January 4th, 1858, however, the distension of the venous system was even greater than on his admission.

The larger veins were abruptly dilated at intervals, indicating the situation of their valves. There was distinct pulsation of cardiac rhythm in the veins of the arm; pressure on a vein stopped the pulsation on the distal, but not on the proximal side of the point of pressure; the pulsation, therefore, was not transmitted through the capillaries. The thrill in the veins at the root of the neck was very perceptible to touch, it was synchronous with each beat of the heart. A loud systolic murmur, evidently generated in the veins, was audible at the spot where the thrill was to be felt. There was neither thrill nor murmur in the carotid or subclavian arteries.

At two points, where the bulging of the brachial vein attained the size of half a broad bean, the pulsation of the vein was perceptible to touch as well as eye.

There was orthopnœa, extreme anasarca, and ascites.

After death about eight ounces of reddish serosity was found in either pleura, and ten ounces in the pericardium.

The heart was much larger than natural. The right auriculo-ventricular opening measured six and a quarter inches in circumference. The left auriculo-ventricular opening was so much contracted from old disease of the mitral valve, that even the points of two fingers passed through it with difficulty. All the cavities of the heart were much dilated; that of the left ventricle, however, less so than the others.

The walls of the heart were thicker and much harder and tougher than natural. When cut across they did not fall inwards, but the cavities still retained their rounded form unchanged.

The coronary sinus was very capacious; the right internal jugular vein was enormously dilated; the valves at its orifice were perfect, as were the valves of the veins of the arms.

There was no disease of the aorta itself, and only adhesion to a trifling extent of two adjacent aortic sigmoid valves to each other.

The aortic orifice measured three inches in circumference. The pulmonary artery measured three inches and a half in circumference.

The liver was small, but singularly tough ; it was strongly granular; the tissues of the portal canals were especially thick and tough; the acute margin of the organ was reduced to fibro-cellular tissue. The kidneys were exceedingly hard and tough, and finely granular. The lungs were the seat of pulmonary apoplexy and congestive pneumonia.

Remarks.—The starting point of the disease of the heart in this case evidently was at the mitral orifice; to this all the other diseased states of the organ were mechanically referable. The disease of the mitral orifice was doubtless, notwithstanding the history, of long standing.

How extreme must have been the impediment to the passage of the blood through the right side of the heart, was shown by the remarkable fulness of the superficial veins. The distension of the right jugular vein was so great that the valves at its opening into the subclavian vein no longer sufficed to close the aperture, and regurgitation was permitted; in the same way as regurgitation was permitted through the dilated right auriculo-ventricular orifice from insufficiency of its healthily sized valves to close the abnormally large aperture. The thrill felt at the root of the neck, and the systolic murmur heard at the same point, were evidently due to the same cause, viz., to the flow of blood past the margin of the valves into the dilated jugular vein.

That the same cause which had led to the distension of the visible veins, viz., the impediment to the escape of blood from the right side of the heart, had also led to distension of the veins of the heart, was shown by the dilatation of the coronary sinus and the amount of serosity in the pericardium. The condition of the liver and kidneys indicated that they were the seat of the exudation of contractile lymph, favoured by, if not the direct result of, their mechanical congestion. The induration and the toughness of the heart were so great that the attention was at once arrested by them.

Case.—T. B—, æt. 9. This boy had from a very early period of his life been the subject of well-marked cyanosis, and some time before death suffered from a considerable amount of anasarca.

After death, in the *pericardium* was found rather more than two ounces of transparent serosity, and its tissue was more opaque and thicker than natural. Here and there beneath the visceral pericardium were crimson spots due to extravasation of blood; there was a good deal of serosity in the loose cellular tissue at the base of the heart, *i. e.* between the auricles and ventricles.

The *right auricle* and *venæ cavæ* were filled with recent clots and fluid blood. In the appendix of the auricle was an old clot, and a clot also of some age was interlaced among the columnæ carneæ of the *right ventricle.*

The *left auricle* contained much fluid blood, and in its appendix was an old clot.

The *left ventricle* was almost filled with old clot; at first sight it looked as if a fungous growth was sprouting upwards from the apex. A recently formed clot extended from the old clot into the aorta.

The right and left sides of the heart, both auricles and ventricles, were greatly dilated, and their walls thickened. The *valves* were quite healthy.

Dimensions of orifices—aortic, $1\frac{7}{8}$ths inch; pulmonary artery, $2\frac{1}{2}$; left auriculo-ventricular, $3\frac{1}{8}$th; right auriculo-ventricular, $4\frac{1}{8}$th. The weight of the heart, with the old clot in the left ventricle, $7\frac{3}{4}$ ounces.

The *coronary sinus* was of very large size.

The liver, spleen, and kidneys were all remarkable for their hardness and toughness.

As far as could be ascertained this boy had been the subject of cyanosis from his earliest infancy. The long duration and the extreme degree of the impediment to the circulation through the lungs and right side of the heart were shown by the large size of the pulmonary artery and tricuspid orifices and of the coronary sinus, as well as by the state of the liver and kidneys. That there was no great cardiac impediment to the circulation at the time of birth was proved by the closure of the foramen ovale. The sub-pericardial hæmorrhage, and the œdema of the cellular tissue at the base of the heart, indicated a sudden extreme increase in the obstacle to the circulation.

Remarks.—It is probable that the primary affection was endocarditis of the apex of the left ventricle occurring very soon after or shortly before birth; that coagulation of blood on the roughened surface followed, and that to the clot so formed the impediment to the circulation was due. The enlargement, hardness, and toughness of the abdominal viscera evidently resulted from their mechanical congestion. Can we doubt that the extreme induration and toughness of the walls of the heart were due to the same cause?

Case.—J. R—, æt. 47, a cabman, of intemperate habits, was admitted into University College Hospital, November 17th, 1857, suffering from extensive and extreme hypertrophous emphysema of both lungs, capillary bronchitis, and hypertrophy and dilatation of the heart, more especially affecting its right side.

The man stated that he had been for many years subject to winter cough, and that he had previously had five or six attacks of bronchitis, the last about a year ago; that his breath was always short, and his face and lips dusky purple in colour; that a year since he first observed that his legs were swollen, and shortly after that his abdomen was larger than natural; that he vomited a little blood two years ago, had often coughed up mucus streaked with blood, and had suffered repeatedly from epistaxis.

He dated his present illness from the 9th of November, when he had rigors, followed by heat of surface, thirst, increase of cough, and dyspnœa, and was obliged to take to his bed.

When I saw him after he entered the hospital he was suffering from extreme orthopnœa. The whole surface was livid. The vessels of the conjunctivæ were dilated and filled with blood; the eyeballs were prominent, the lower lip swollen and everted. The large veins of the neck, which during inspiration were scarcely perceptible, were distended during expiration, but did not pulsate. The heart's sounds were not audible, though its pulsations were perceptible below the ensiform cartilage. There were present all the physical signs of extreme, extensive hypertrophous emphysema. There was some anasarca. The pulse was 132, very small and weak; the respirations were 38 in the minute. The urine contained a large quantity of albumen, and some small waxy casts.

On the 18th of December the colour of the man's face, trunk, and extremities was darker, *i. e.* he was more cyanosed, than I ever saw the skin of a patient who was not the subject of congenital heart disease.

The lividity varied in degree during the succeeding fortnight. On the 28th the veins of the neck were constantly much distended, and pulsated; there was a large quantity of fluid in the peritoneal cavity; the anasarca had increased in amount; there was a systolic murmur audible at the base of the sternum, *i. e.* just above the ensiform cartilage.

On the 30th my notes were—Cyanotic symptoms more marked than at any time since admission. Heart's beat lower. Left lobe of the liver considerably depressed by the manifestly over-distended right side of the heart and the emphysematous lungs. Veins of neck extremely full, knotted, pulsating.

The man died on the 31st.

On examination after death there was found extreme hypertrophous emphysema of both lungs; great hypertrophy and dilatation of the right side of the heart, moderate hypertrophy and dilatation of the left side. The walls of the heart were indurated and very tough; this change affected the right side infinitely more perfectly than the left. The muscular tissue of the right side of the heart was very compact, and the cut surface particularly smooth and homogeneous in appearance. The mitral orifice measured 4½ inches in circumference; the tricuspid, 5½ inches; the aortic, 3 inches; the pulmonary artery, 3½ inches. The coronary sinus admitted the little finger with facility; it was evidently much dilated. There was general anasarca, and a considerable amount of fluid in the peritoneal cavity.

The liver was enlarged, it weighed fifty-two ounces. It was uniformly and finely granular; it was hard and very tough. The kidneys were large and irregularly contracted, so as to be coarsely, unevenly granular on the surface. They weighed six ounces each.

Remarks.—This case affords a good example of the most common causes of over-distension of the right cavities of the heart, and therefore of congestion of the walls of the heart, and, as a consequence, of induration, toughening, and permanent dilatation of the heart; viz., repeated attacks of bronchitis and hypertrophous emphysema. The congestion of the whole venous system showed the great impediment that existed to the flow of blood through the right side of the heart; and the extent to which the capillary system had suffered dilatation proved the length of time that the impediment had existed. The anasarca and the large size of the coronary sinus supported the same inferences.

The liver and kidneys were all enlarged, hard, and tough —changes doubtless chiefly due to their long-continued mechanically produced congestion. The fluid in the peritoneum and the albumen in the urine were also, probably, due to the obstacle to the return of blood from the liver and kidneys. The induration of the heart was very decided; the specific gravity of the most markedly indurated part of the right ventricle was taken for me by Mr. Russell, in Professor Williamson's laboratory; it was 1·053.

The very trifling evidence of old pulmonary collapse found after death, and the enormous extent and degree of the emphysema, conjoined with the fact that, during life, there was very forcible protrusion of the intercostal spaces above the level of the ribs, and equivalent prominence of all the soft parts of the thoracic parietes in violent expiration, told strongly in favour of the expiratory theory of emphysema laid before the Society by myself in 1857.

Case.—Robert W—, aged two years, was a healthy child till the commencement of 1858, when, his mother said, he suffered from bronchitis. During that attack she noted that his feet were swollen. All œdema disappeared in the course of the ensuing spring, but he never recovered his previous state of health.

The child was admitted into the Hospital for Sick Children on January 30th, 1859—his mother stating that his present illness was of only three days' duration.

There were present the physical signs of general but imperfect solidification of both lungs; strongly marked pulsation in the jugular and subclavian veins; a little lividity of the lips; œdema of legs, hands, and face. The child was seized with convulsions on the 1st of February, and died.

After death the whole of both lungs were found to be loaded with gray granulations and yellow tubercles; and here and there, in addition to much collapse, were nodules of lung solid from pneumonia.

About two drachms of serosity were found in the pericardium; the veins on the surface of the heart were filled with blood.

The pulmonary veins, the venæ cavæ, both auricles, the right ventricle, the pulmonary artery, and the aorta contained a large quantity of dark, loosely coagulated blood. The walls of the right ventricle were only slightly thicker, but were much tougher and harder than natural. The walls of the left ventricle were much less tough and hard than were those of the right ventricle, and its cavity was not dilated.

Examined by the aid of the microscope, the muscular fibres of both ventricles were found to be studded within and without by protein-granules. A few of the muscular fibres were in a state of fatty degeneration. The most completely degenerated fibres were found in the muscular band which passes from the right wall to the septum ventriculorum.

The spleen, liver, and kidneys, were all loaded with blood, and tougher than natural.

Remarks.—Here the primary impediment to the circulation of the blood was seated in the lungs. Bronchitis, pulmonary collapse, emphysema, and an enormous accumulation of tubercles in every part of the lungs, were the causes of the obstacle; the degree and duration of the impediment were indicated by the dilatation of the right auriculo-ventricular orifice, by the state of the spleen, of the liver, of the kidneys, and of the veins, and by the anasarca.

The texture of the heart was damaged in the same mode and from the same cause as was the texture of the liver, spleen, and kidneys, *i. e.* from mechanically produced venous congestion.

Case.—E. B—, aged eight years and nine months, was admitted into the Hospital for Sick Children on September 10th, 1859. In November, 1856, she suffered from severe rheumatic fever, and from that time had been obliged to cease her occupation, viz., that of a tight-rope dancer. When she came under my care she was suffering from extreme anasarca, ascites, great fulness and pulsation of the veins of the neck and upper extremities, and orthopnœa. The chest was in form the type of that which accompanies great hypertrophy and dilatation of the heart in a child. The physical signs indicated very plainly the lesions found after death.

She died October 3d, 1859; the body was examined twelve hours after death. The sternum being removed, the tissues of the mediastinum were found to be much more vascular than natural, and, in common with the cellular tissue at the root of the neck, were infiltrated with serosity, and remarkably tough. There were about four ounces of transparent serosity in the pericardium. Numerous crimson spots, due to sub-pericardial extravasation of blood, were present at the base of the heart, especially about the root of the pulmonary artery; the loose cellular tissue between the ventricles and auricles contained much serosity. Both auricles contained very large coagula; that in the right auricle was continuous with a clot in the coronary sinus. The latter clot, when removed from the sinus, measured three inches in length. The veins on the surface of the heart were gorged with dark-coloured blood. The walls of the heart were greatly hypertrophied, especially those of the right ventricle; they were remarkably hard, tough, and dark in colour. The tissue of the walls was more compact, its cut surface smoother, its edges sharper, and its aspect more transparent than natural. All the cavities of the heart, excepting that of the left ventricle, were extremely capacious. The left auriculo-ventricular orifice was greatly reduced in size, from disease of the mitral valve; the right measured $3\frac{3}{8}$ths inches in circumference; the aortic orifice, $1\frac{7}{8}$ths inch; the pulmonary artery, $2\frac{1}{2}$ inches.

The liver, spleen, and kidneys were all loaded with blood, and very tough.

Examined by the aid of the microscope, the muscular fibres of the heart were found to be paler, tougher, and more closely united to each other than natural; they were covered with very fine molecular granules; their transverse striæ were very imperfect. There appeared to be an excess of fibrous tissue in the walls of the heart.

Remarks.—The primary disease in this case was evidently constriction of the mitral orifice, and the duration of the disease three years. The toughness, &c., of the cellular tissue, the state of the liver, spleen, and kidneys, the dilatation of the pulmonary artery, the serosity in the pericardium, the fluid in the cellular tissue at the base of the heart, the dilatation and engorgement with blood of the coronary sinus and the veins on the surface of the heart, and the sub-pericardial extravasation of blood, were all evidently due to mechanical impediment to the passage of the blood through the left auriculo-ventricular orifice.

The change in the texture of the heart was, as in the preceding, the result of the same cause that produced the change in the texture of the liver, spleen, and kidneys; and the change was of the same nature; only, in the case of the heart, the call on the organ to act was more powerful than in health, and the impediment to the onward flow of the blood necessarily induced over-distension of the cavities behind the impediment.

CASE

OF

ACUTE CARIES OF THE WALLS OF THE TYMPANIC CAVITY,

PRODUCING

ULCERATION OF THE INTERNAL CAROTID ARTERY.

BY

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AURAL SURGEON TO ST. MARY'S HOSPITAL, AND LECTURER ON AURAL SURGERY AT ST. MARY'S HOSPITAL MEDICAL SCHOOL.

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The cavity of the tympanum is remarkable for its intimate relations with important organs. Thus, above it, lie the dura mater and the middle lobe of the cerebrum; below it is the jugular vein; in front is the internal carotid artery; and behind it are the lateral sinus and cerebellum. Internally are the portio dura nerve and the labyrinth; while through the tympanic cavity itself passes the chorda tympani nerve.

When it is borne in mind that this cavity communicates with the outer air, through the Eustachian tube, and that it is protected externally only by the delicate membrana tympani, it will not be a source of surprise that diseases of the mucous membrane of the tympanum are very prevalent, nor that, when they communicate with neighbouring structures, they frequently terminate fatally.

It is known full well that disease often extends from the tympanic walls upwards to the dura mater and cerebrum, and backwards to the lateral sinus and cerebellum; it sometimes advances downwards to the jugular vein, but it is extremely rare for the internal carotid artery to be seriously implicated. Indeed, with the exception of the case to which I shall subsequently refer, and which was cited by Professor Hewett, in his lectures at the Royal College of Surgeons, in the year 1859, I am not aware that any instance has been reported of hæmorrhage from the internal carotid artery, originating in caries of the petrous bone.

If we inquire into the cause of the rarity of ulceration of the internal carotid artery, a satisfactory answer may probably be found in the fact, that caries of the osseous walls of the tympanum is usually a chronic disease; that it commonly results from secretion being pent up in the tympanic cavity, and that this secretion accumulates in largest quantities in the middle part of the tympanum (remote from the position of the Eustachian tube and carotid canal) and presses upon the delicate upper wall of the tympanum, which easily yields. On the contrary, the case of caries of the canalis caroticus, already on record, and the one I am about to relate, appear to have been instances of acute disease occurring in the mucous membrane and in the bone, and wholly independent of any accumulation of secretion.

Case.—W. C—, æt. 46, a jobbing builder, was admitted under my care at St. Mary's Hospital as an out-patient, on the 18th of August, 1859. He was sallow and thin; he had a careworn aspect; his pulse was quick and weak. He had long complained of pain in the left side of the chest, and was reported to have undergone much domestic trouble, which had undermined his health. Two years before I saw him he received a violent blow on the head, above the left ear, producing a wound which bled profusely at the time, but soon healed. Three months previous to his admission at the hospital, during a violent fit of coughing, he was seized with intense pain in the left ear, which continued, with scarcely any intermission, to the time of my seeing him. At the expiration of a few days from the attack of pain, a copious discharge took place from the ear, which has never ceased. At the time of my seeing the patient, the discharge was of a muco-purulent character, and it had a peculiarly sickly and fetid odour, characteristic of diseased bone.

On making a careful examination, the dermis lining the meatus externus was observed to be much tumefied, and no portion of the membrana tympani could be detected. The pain which the patient most complained of was described as being situated very deep in the ear; it was constant and lancinating: he also complained of tenderness below the ear and down the neck, in the course of the large blood-vessels, where there was some degree of tumefaction.

The treatment consisted in the application of leeches below and of blisters behind the ear and at the nape of the neck, in opiate injections, and fomentations. Morphia, aconite, quinine, steel, and iodide of potassium were administered in succession, but neither local nor general treatment appeared to have the slightest effect upon the progress of the disease; the only relief that the patient obtained was when under the influence of large doses of morphia. The symptoms continued without change till the 10th of January, 1860, when the incus came away in the discharge. It did not show any signs of disease, except that the inferior extremity of its long process was rough, and the os orbiculare had disappeared. When I saw the patient on the 16th of January, he stated that during the previous two days, he had twice in the twenty-four hours observed blood of a bright-red colour to pass out of the ear for four or five minutes; in the intervals of the hæmorrhage the pain had been excessive, and the discharge copious and fetid. Upon examination, there was no appearance of blood in the tympanic cavity, the mucous membrane having a pulpy appearance. From the fact of the blood being of a bright-red colour, and from the rapidity with which it flowed from the ear, it was evident that the internal carotid artery had become involved in the disease of the tympanum, that the blood escaped from its cavity, and, most probably, through an ulcerated opening. A consultation was accordingly held with my colleague, Mr. Ure, who proposed, should the bleeding recur, to tie the carotid artery. The patient, however, resolutely declined to become an inmate of the hospital. It was consequently decided that, if the bleeding recurred, pressure on the carotid artery, and plugging the external meatus, should be resorted to, and five-grain doses of gallic acid were administered thrice daily. The hæmorrhage returned on the following day; the plugging of the external meatus arrested it for a time, but subsequently it took place from the nose. The neck of the patient was so extremely tender, that he could not bear any pressure on the carotid.

As the patient now sent for my friend, Mr. Charles Aikin, who resided near to him, and who had attended him previous to his admission as a patient of the hospital, and as he still refused to become an in-patient, he gave up his letter, and I promised to continue my attendance as before, in conjunction with Mr. Aikin.

On the 18th of January, the hæmorrhage having recurred in considerable quantity, Mr. Lane, who was in attendance at the hospital, was called into consultation. The patient's strength was now greatly reduced by the effects of the disease, the intense and prolonged pain he had undergone, and by the loss of blood; and, it being evident that active and destructive disease was making rapid progress in the ear, all hope of benefit from the operation of tying the carotid artery was abandoned, and attention was directed to the alleviation of the pain. To this end, large doses of morphia were administered at frequent intervals, which afforded relief; slight hæmorrhage came on every eight or ten hours, and the patient gradually sank, and died on the 27th of January.

Autopsy.—Large tuberculous cavities were found in each lung.

No disease was detected in the cerebrum, cerebellum, pia mater, or dura mater.

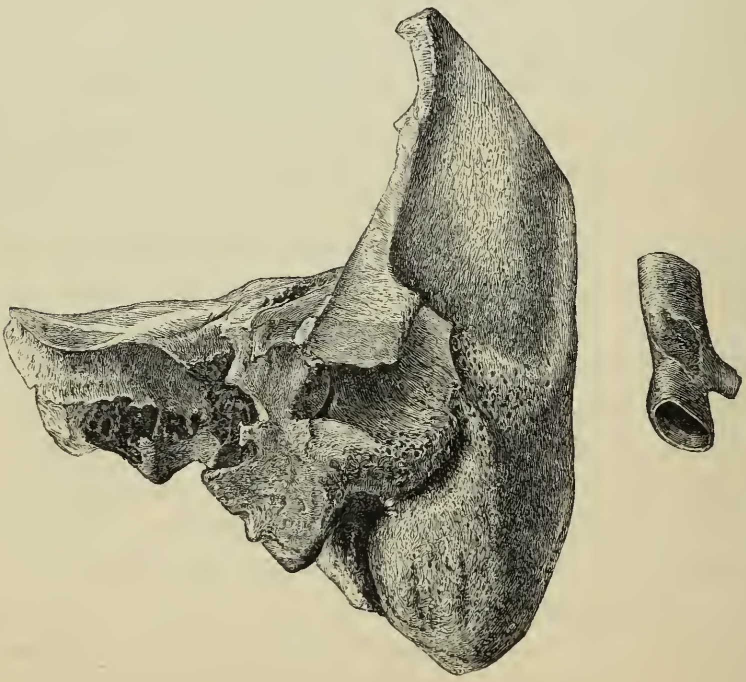
A considerable quantity of dark-coloured purulent matter was found in the upper and anterior part of the neck ; this was continuous with an orifice in the external meatus, through which pus escaped and percolated downwards. This opening in the external meatus was situated within half an inch of the orifice: it was at the lower part, and admitted an ordinary-sized probe. The dermoid meatus was much thickened, but not ulcerated at any other part. The membrana tympani had wholly disappeared, as well as the malleus and incus ; the stapes was detached from the fenestra ovalis and from the tendon of the stapedius muscle, and was free in the tympanic cavity. The mucous membrane had been removed by ulceration from the whole of the surface of the promontory, which was bare ; and at the posterior and inner part of the tympanic cavity were small, rounded, polypoid growths, covering carious bone. The upper osseous wall of the tympanum was destroyed, and there was an orifice communicating with the middle cerebral fossa, sufficiently large to admit a small horse-bean. The upper wall of the horizontal portion of the mastoid cells was also carious, and presented numerous minute orifices. The posterior wall of the mastoid cells formed by the sulcus lateralis was also carious, and exhibited several small apertures. The lateral sinus contained a clot of fibrine, but no pus. The dura mater covering the orifices in the tympanum and mastoid cells was somewhat thicker than natural, and lymph was effused on its surface looking towards the bone. The inferior part of the anterior wall of the tympanum was almost wholly destroyed by caries, so that it and the cavity of the canalis caroticus were directly continuous by means of several large apertures ; the inner wall of the canalis caroticus was also carious. Where the internal carotid artery was in contact with the carious wall of the tympanum, it presented an aperture about two lines in diameter, through which the blood had escaped during life into the cavity of the tympanum. The borders of this aperture were soft and tumefied. The lower wall of the tympanum, separating it from the fossa jugularis, presented several small carious orifices. The cellular structure of the petrous bone between the meatus auditorius internus and the carotid canal was soft and carious.

The question which presents itself to the mind of the Surgeon who hears the particulars of this case is—could no other steps have been taken to save the life of the patient? Every effort that was made with the object of arresting the progress of the caries was of no avail; and there is, I think, but little doubt that, if the carotid artery had been tied with the view of arresting the hæmorrhage, the disease would have advanced, and probably with greater rapidity. Although the loss of blood doubtless hastened the death of the patient, its principal cause was the exhaustion produced by the intense pain, and by the rapid extension of the disease; and, supposing no hæmorrhage had occurred, life could scarcely have been prolonged more than a few days.

The case cited by Mr. Hewett is reported in the second volume (third series) of the 'Archives générales de Médecine,' 1837, by M. A. Boinet, then *Interne* of the Hôtel Dieu of Paris: it is in some respects singularly analogous to the one just related. I may perhaps be permitted to give some of the leading facts. In the year 1834, a man, æt. 42, applied at the Hôtel Dieu on account of a purulent discharge from the left ear. In 1826, while tipsy, he was brought to the same hospital, having fallen from a fourth story. There was a wound at the upper and back part of the head, with a depression of the cranium. Small portions of bone were extracted, and he left the hospital after a sojourn of two months. About six years afterwards, without any known cause, noises came on in the left ear, accompanied by acute pain which extended over the head; a discharge speedily followed. Received into the Hôtel Dieu again, he was bled locally and generally, and blistered, and a seton was applied at the nape of the neck. Under this treatment the discharge diminished, and he left the hospital after a sojourn of five months. He continued his occupations for about a year, when he again returned to the hospital, with an increase of pain and discharge. About a month after his stay in the hospital, he was seized with paralysis of the left side of the face; the other symptoms abated, and he again left the hospital, but returned at the end of two years and a half. He complained of a copious discharge from the left ear, but not of pain in the head. Two months after he had been in the hospital there was a sudden rush of red blood from the mouth, nose, and left ear; the hæmorrhage lasted for ten minutes. On the following morning the hæmorrhage recurred, and again arrested itself, without quite so much loss as on the previous day. In the evening violent hæmorrhage occurred, which it was found impossible to arrest, and the patient died from loss of blood.

Autopsy.—The substance of the brain was firm, and rather pale than otherwise. The cerebellum was healthy. The dura mater presented a slightly blackish tint at the posterior part of the petrous bone, and also at its base in front; on removing this membrane a roundish aperture, three lines in diameter, was observed in the upper surface of the petrous bone. There was also a large aperture at the lower part of the petrous bone, the whole of the jugular fossa having been destroyed. The styloid process was detached from the rest of the bone. The cochlea and the semicircular canals were laid open in parts; the orifice of the Eustachian tube and the carotid canal had completely disappeared. The mastoid cells and the mastoid process were in a natural state. The portio dura nerve, from its entrance into the aqueduct of Fallopius to its passage behind the mastoid process, was destroyed. The left sinus lateralis was almost obliterated at its lower part, while at its upper part there was a clot adhering to its surface, but without any traces of pus. The internal carotid artery, of which the coats were very thin and slightly soft, presented an ulcerated opening, about a line in diameter, at its anterior part and about the middle of its course in the temporal bone. It did not contain any clots.

A preparation from my Museum, which I lay before the Society, reveals a state of parts very analogous to that described by M. Boinet, with the exception that the disease had extended to the lateral sinus, and not upwards. The preparation was presented to me, and I regret that I am unable to give any particulars of the case, as it is very rare that so extensive disease is met with. The whole of the meatus externus and mastoid process have been removed, the styloid process detached, the entire osseous sulcus lateralis in the temporal bone absorbed, the lower wall of the tympanum destroyed, as well as the fossa jugularis, and the posterior part of the canalis caroticus.



Carious temporal bone and ulcerated internal carotid artery.

ON

DISEASES OF THE KIDNEY

ACCOMPANIED BY

ALBUMINURIA,

CONSIDERED IN RELATION TO

THEIR ORIGIN IN CHANGE OCCURRING IN THE TUBES OR IN THE INTERTUBULAR STRUCTURE.

BY

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SEVERAL years ago I collected some evidence upon the subject of Bright's disease, believing that the kidney which was once large, mottled, and glossy, required nothing but time to become shrivelled and granular. I soon met with facts which compelled me to renounce this opinion, and to assume that the two conditions involved quite different changes. Believing that morbid anatomy must be the foundation of all sound pathology, I have since applied myself to the minute examination of the kidney under its various aspects of health and disease, and the results I have obtained will form the basis of the present paper.

I believe I shall be able to show that all those conditions of kidney which have been called after the name of Dr. Bright, whether rough or smooth, large or small, whatever their external appearance may be, are capable of arrangement into two great classes; that in the one we have disease proper to the secreting surface of the tubes, while in the other the disease is essentially intertubular; and that the great characteristic difference between these two morbid conditions is, the absence or presence of superficial granulation.

I will commence by endeavouring to explain the pathology of the former of these complaints, where the surface of the kidney remains even. This I will venture to call the—

Tubular Disease.—I shall include, in a general description, all the varieties of the large smooth kidney, regarding as unimportant all those distinctions which depend on the quantity of oil which happens to be in the epithelial cells, or of fibrinous matter in the tubes.

This state of kidney has been well explained by Mr. Simon, in a paper read before this society. In the progress of the disease may be recognised two distinct stages. First, the period of enlargement; secondly, the period of diminution.

But, before undertaking to describe the minute anatomy of either of these conditions, I may be allowed to explain the methods I have used in the investigation. Besides the ordinary plan of tearing the organ into shreds with needles, and looking at the component parts under a high power, it has been my habit to prepare translucent sections of the gland, made in such a way as to show the tubes and the Malpighian bodies in their living relation to each other, and to the intertubular material. A piece of the kidney is boiled in water, slightly acidulated with acetic acid; it is then hung up to dry, and in a short time acquires much of the appearance and texture of gutta percha. If a smooth surface be now cut, and then moistened with a drop of water, it will be found that, from this damp surface, sections can be obtained of any degree of tenuity, which will present a beautiful picture of the anatomy of the kidney. These require a magnifying power of from 80 to 100 diameters. Plate VII is a good representation of such a section, taken from a healthy kidney.

Much that I shall set forth has been deduced from examinations conducted in this way. A person who wishes to obtain a knowledge of the structure of any organ, may spend his life in picking it into small pieces, and looking at the component parts under a high power, without ever being able to do more than enumerate the elements of which it consists. His microscopic eye cannot appreciate the relationship of the several parts. It is essential that some method should be devised which shows more of the organ as a whole; in its habit as it lived; with its elements bearing their natural relation to each other. Now I think I shall be expressing the conviction of many of my readers, when I say that ordinary dexterity is quite insufficient to obtain a section of the fresh kidney which answers this purpose; but when prepared in the way I have described, the section is made so easily, and is so clear, that nothing remains to be desired.

Returning now from this necessary digression, I will endeavour to unfold the pathology of the large smooth kidney. From the commencement of the disease, the gland gradually increases in bulk; its weight is often more than doubled. Sometimes in the earlier periods a certain amount of congestion is visible, but this soon becomes masked by the opaque epithelium which is the cause of the enlargement. The characteristic aspect of bloodlessness, which belongs to the disease, may be produced in as short a time as four days. I shall presently show that this anæmia is more in appearance than in reality, and that excessive secretion is the essence of the disorder. If the complaint has, as yet, made but little progress, there will be seen on section a coarseness of texture, an appearance as of two materials, a red and a buff, roughly intermingled, and often sprinkled throughout with red points, which are injected Malpighian bodies.

As the disease progresses, the appearance becomes more characteristic. The surface invariably remains perfectly smooth, and the capsule loose and thin as in health. When this investment is removed, little or nothing is seen of the superficial capillaries which should divide the surface into lobules. There is great apparent want of vascularity, but here and there may be seen a patch where red vessels are visible on the surface, and the kidney is accordingly described as mottled. When cut open, it is seen that the cortex is greatly increased in bulk; the cones usually being enlarged too, though seldom to a corresponding extent. The cortical substance varies much in appearance, according to the nature of the secretion which distends the tubes. It is sometimes, particularly when the disease has followed scarlet fever, almost like ivory in colour and fineness of texture; generally it has a yellow tinge and coarser grain, and resembles the cut surface of a parsnip. In the former case the tubes will be found stuffed with healthy epithelium; in the latter, the cells have undergone more or less fatty degeneration. The cones sometimes retain their ordinary appearance, which is exaggerated by contrast; usually they are paler than in health, owing to the same sort of change as has affected the rest of the organ. Such are the appearances which may be observed by the unassisted eye in well established cases of the tubular disease. I will endeavour to explain the elementary changes to which they are due.

If we examine a section of a kidney in this condition, prepared in the way I have already advocated, we shall find the organ anatomically complete. The tubes are all entire and everywhere in contact with each other; they may appear a little swollen, but with the low power used this change is not very manifest. The Malpighian bodies are everywhere at the usual distance apart, showing that there has been no loss of renal substance; and, on the whole, the drawing No. 1, which is an excellent representation of a healthy kidney, may pass for a portrait of this stage of tubular disease. From an examination thus conducted, our information, is in this instance, of a negative character.

With higher powers and the fresh kidney, we find the disease to be essentially of the tubes. The ducts which compose the cortex are invariably choked up with their own secretion, perhaps here and there discoloured with blood.

The cells may remain entire or may be more or less broken down. They are more apt to lose their integrity when there is a tendency to fatty degeneration. Sometimes nothing can be seen in the tubes but the oil globules, into which the altered cells have broken. In twenty-two characteristic specimens of the disease, it was found that the matter distending the tortuous ducts; was the epithelial secretion of the organ, and that only, save in a solitary instance, where a little fibrinous matter was found. This epithelium is in most cases, more or less fatty, and more or less broken down. Unbroken and unchanged cells were found in seven cases; but in only one of these did they exist to the exclusion of everything else. In sixteen, the ducts contained fatty epithelium, this being generally much disintegrated. Out of the whole number, it appeared that the distension of the organ was due about as much to the presence of comminuted cells, as to that of unbroken ones. The frequency of these changes, as affecting the cortical structure, may be thus stated :

Table showing the contents of the convoluted tubes in twenty-two cases of large smooth kidney.

Entire cells alone in 4	These containing oil in 3. Not containing oil in 1.
Entire and broken cells together in 15	With oil in 12. Without oil in 3.
Broken cells alone in 2	With oil in 1. Without oil in 1.
Fibrinous matter in 1	With oil and broken cells.

A fatty condition of the epithelium is very much more common with the smooth kidney than with the granular. It is probable that it is a consequence rather than the cause of the derangement. Possibly the tube membrane is altered by the inflammation or congestion which constitutes the disease, and is rendered apt to produce cells slightly erroneous in composition, so as either to contain oil originally, or to decompose into oil soon after their detachment.

I have observed that when the disease has followed scarlet fever, there is little tendency to this degeneration; but when it has come on in consequence of a definite exposure to cold, then, almost invariably, oil forms the bulk of the tubular contents. In other organs, as in this, oil often appears as the result of inflammatory action. If we peel the layer of fibrine from the surface of a heart affected with pericarditis, the superficial muscular fibres often appear to be transformed into oil globules, while those placed deeper remain in their natural condition. Similarly oil is developed in the lung under pneumonia, and probably in the brain under the influence of inflammatory softening; but it is not necessary to multiply examples of a sequence which is familiar to all pathologists. Such facts show the likelihood that "fatty degeneration of the kidney" is not a specific and independent disorder, but is merely an occasional accompaniment of the tubular disease, which essentially consists in increased epithelial growth, depending on inflammation or some allied condition.

In the later stages of this disease, a considerable quantity of pus is sometimes found in the urine, which has obviously been secreted by the tubes. Pus globules are seen imbedded in the casts, and after death the same product is found in the ducts. In one and the same case may be seen sometimes all the intermediate links by which the normal epithelium has become thus altered.

Whatever be the state of the kidney, whether smooth or granular, cells are occasionally observed in the urine, which at first look like natural glandular epithelium, but under the action of acetic acid exhibit the compound nucleus characteristic of pus. But in the smooth kidney alone is this secretion ever found in sufficient quantity to attract attention. It here sometimes forms a deposit obvious enough to the unaided eye. In the granular kidney, the only trace of its existence consists in a few scattered cells only to be recognised by microscopic research, and after subsidence.

With regard to the breaking up of the epithelium, I may observe that the integrity of the cells is the measure of the acuteness of the disease. Where that secretion has lost its form, it may be presumed to have remained long in the tubes. In five well-marked cases of the disease, where the distending material was almost entirely unbroken epithelial cells, the duration of the symptoms had varied from three weeks to four months. In the same number of cases, where the secretion was chiefly comminuted, the disease had endured for periods varying from three months to thirteen years. In the former class the average duration of the disorder was less than ten weeks, in the latter it was more than three years. These statements are of course drawn entirely from the examination of the organs after death; but the rule deduced can be easily applied during life by examining the sediment thrown down by the urine. This part of the question, however, is foreign to my present purpose.

A few words upon the state of the tubes in the cones will conclude what I have to say upon the anatomy of the large smooth kidney. These channels being straight, and comparatively short, are little liable to become blocked up.

The products of disease, whether formed within their own cavities, or brought from afar, find a ready exit. Hence it is that the epithelial secretion, which is filling up and spoiling the tortuous ducts, is never present in the same quantity in these, and is frequently absent altogether. It is not unusual to find most of the tubes here perfectly healthy. It appears, however, that generally there is the same tendency to perverted secretion as elsewhere. The morbid product is washed from the surface, and the membrane is afterwards found perfectly bare. Thus, the straight portion of the duct is often in an opposite condition to the convoluted. Instead of being permanently obstructed and impervious, it is continually becoming filled up with a glossy fibrinous material, which is repeatedly shed in the form of cylinders. Probably, owing to some change in themselves, the interior of these casts becomes generally studded with specks of oil—or cells of epithelium, or blood-corpuscles, which chance to be travelling downwards, may become imbedded. The epithelial product may be in such large quantity as to form the bulk of the cylinder. There can be no doubt that it is mainly in this portion of the gland that casts are produced; for in cases where, for long before death, these productions have all been transparent and glassy, it is very common to find the cortical ducts all choked with coarse, opaque secretion; while in the cones alone is found abundance of the material of which the casts are formed. Besides this, I think we may fairly conclude that in this complaint the cones undertake part of the secreting duty of the organ. The convoluted portion of the tubes are seen crammed with stale secretion, so that they must be perfectly impervious. The casts which were shed, perhaps, may have contained nothing but smooth, glassy fibrin, so that none of the opaque epithelial product could have escaped; and the tubes so circumstanced must have been devoid of any power of secretion. This conviction is further borne out by an examination of the cones themselves; for, in such cases, not only are they frequently of greater extent than usual, as if they had undergone some sort of compensating hypertrophy, but their epithelium is sometimes obviously changed in character, it becomes fuller and more rotund, like that proper to the cortical structure.

Before quitting this portion of my subject, I must briefly notice an unusual form which the disease sometimes assumes. The surface of the gland, instead of being nearly uniform or mottled, so that the shades run into each other, is speckled all over with little, sharply defined, white spots, which look like something foreign to the substance of the organ. These are not, as at first they appear, intertubular, but are little families of tubes, densely filled with fatty epithelium; the ducts between them being affected to a much less degree, or even remaining natural. This condition, however, is so rare as not to demand further discussion.

I will now proceed to sketch what I believe to be the second stage of the tubular disease.

We have traced the progress of the *renal catarrh*, if I may be allowed the expression, from its commencement, in excess of epithelial growth, to its climax in total obstruction of the cortical tubes. The next step is a simple one. The over-stretched tubes, one after another, burst, and their contents, no longer secluded by a surrounding membrane, are brought within the ready operation of the absorbents and are removed. This, however, is a matter of inference rather than of demonstration. Examining in section a number of enlarged smooth kidneys, I found that about half of them had thus commenced to pass from the stage of enlargement to that of decrease, although there was nothing in their outward appearance to indicate it, their bulk still being excessive. A section, taken from a kidney in this state, is represented in the drawing No. 2. The pair weighed sixteen ounces. In such preparations we find places, especially near the surface, where the Malpighian bodies, which have now become enlarged, and enveloped by condensed fibrous tissue, lie in unnatural proximity. Often several almost touch each other. Their intervals are occupied by the debris of collapsed and ruined structure. Shrivelled remnants of the tube-membrane are generally clearly seen. This condition is as yet only partial. A distended duct is often seen winding among the wasted remains of its companions. The tubes, one after another, collapse until a great portion of the once enlarged gland has been reduced to the bulk of little more than its Malpighian bodies. The appearance of the organ affected has by this time undergone some alteration. It has lost its excess of bulk, and even shrunk below the natural size; in one case a single kidney weighed only an ounce and a quarter. The capsule has generally become thickened to a slight degree, and become more disposed to adhere than formerly. The surface remains perfectly even, but often presents to the eye a fine, sandy texture. The colour has undergone but little change. It is still pale, greyish, and apparently wanting in blood. When the organ is cut open, it is manifest that it is chiefly composed of cone substance; an edge, perhaps, no thicker than a shilling, being all that separates that structure from the capsule. What remains of the cortical material is coarse in texture, pale and firm.

The condition I have just described, that of the smooth, dwindled kidney, is seldom attained; though, as I said before, the process which leads to it has been commenced in about half the cases of large, smooth kidney which reach the dead-house. During ten consecutive years, 2350 bodies were examined in St. George's Hospital, and this condition of the renal organs is described only in twelve instances.

A magnified section of a kidney, where this change was fully developed, is represented in plate No. 3; it is a step beyond what was seen before. In such kidneys the peculiarities are well seen by the method made use of. There is close aggregation of the Malpighian bodies; and the fibrous tissue of the organ is condensed around them. The spaces between them are occupied by structure wasted and collapsed, even until it approximates, in some places, to mere fibrous tissue. A few of the tubes are left; and these sometimes intermingle pretty uniformly with the effete remains, and at other times are gathered together into insular masses. The destructive change shows a predilection for the more superficial parts of the organ; it begins at the surface and proceeds inwards. This is what we should expect. A gun barrel bursts near the breech, and the convoluted tube of the kidney gives way at the part furthest removed from its orifice.

Higher magnifying powers and the fresh kidney corroborate these statements, and enable us to add a few particulars. We find that healthy tubes, or, indeed, tubes of any sort, are difficult to meet with; while the mass of the organ presents the appearance of coarse, fibrous tissue, mixed with fibrillating cells, in the midst of which can occasionally be seen natural and shrivelled tubes. There is no reason to believe that there is any unnatural formation of fibrous tissue; this material necessarily forms the bulk of the kidney on the removal of the contents of the ducts. In one of the few specimens of the disease which I have seen, little cysts were so numerous as to give to some parts of the cortex the appearance, under the microscope, of a sponge. The cones were similarly affected, but to a less degree. The arrangement and size of these cysts were such that they could scarcely have been produced by division of the tubules. They were placed rather in nests than in lines, and some were no more than $\frac{1}{1430}$ of an inch in diameter. It seems that the theory put forth by Mr. Simon here holds good. The cysts are developed out of the epithelial germs which are left on the walls of the ruptured tubules. I shall, hereafter, show that under different circumstances cysts are formed in a different way. This brings to an end what I have to say upon the anatomy of the smooth, mottled kidney. I will now pass on to the granular or intertubular disease.

It will be convenient to describe first a most important stage, that of commencement; marking the earliest signs by which the intertubular effusion may be recognised. This, I hope, will tend to throw light upon the subsequent progress of the disease. This done, I shall take leave to divide granular kidneys into two families, and in so doing I shall study convenience rather than strict pathological accuracy; for, though extreme cases are different enough, these are merely the terminal links of an unbroken chain. I may be allowed to speak of these as the chronic and the acute varieties of the intertubular disease.

First, then, as to the period of commencement. It is common to meet with kidneys as to which there is a doubt whether they are healthy or not. The capsule is a little thickened, and it is more adherent than it should be. When removed, the surface presents a confused, muddy appearance, and it may be noticed that a thin layer of capsule is still adhering to the kidney. As yet there is no distinct granulation, though the tendency may be indicated by one or two curved depressions, or by a little general roughness. On examining such a kidney, in section, we shall find all the central parts quite healthy, save that closely attached to the inner capsular film; we shall see certain narrow projections insinuating themselves between the tubes. If we observe the position of an external depression or cicatrix, we shall find that from this passes inwards a process of fibrous-looking material, which is seen to involve in its interior the contracted remains of tubes it has imbedded in its passage. It looks as if an effusion, small in amount, had commenced in the surface and worked its way inwards among the ducts, numbers of them thus becoming completely surrounded. After a time contraction follows in the wake of the exudation, and the tubes imbedded are reduced to mere microscopic threads. At the same time appears another result of the contractile tendency. The points at which the processes originate become depressed; and when these are numerous and tolerably regularly distributed, the result is superficial granulation.

As we daily see, the tubes themselves may be distended to the last degree, the surface of the organ still remaining perfectly smooth and glossy. And we should be at a loss to explain how it should be otherwise. The cortical substance is a uniform entanglement of ducts, twisting through meshes of fibrous network, which are so minute that they are far beyond the range of any eye but the microscopic. And when the swelling of the kidney is limited only by this network, and by the distensibility of the tubes themselves, a surface must be produced which is to the eye and touch perfectly smooth. To produce a puckered or granular surface, we must have something else. It is essential that there should be some means of tying down the surface at numerous points, between which only is expansion possible. This condition is provided in the manner I have endeavoured to explain.

The earlier kidneys are examined in this disease, the larger they are. At the very commencement of granulation I have found them weighing together sixteen ounces. As contraction proceeds they become, as is well known, much below the natural size.

The foregoing observations may be verified upon the prepared section with a low power. If now we resort to the quarter-inch object glass, and examine the tubes at the earliest period at which we can surely recognise the disease, we may find all the tubes perfectly natural, and the epithelium exactly what it is in a state of health. More commonly we shall find some of the tubes healthy, while others are filled with whole or broken down epithelium; this being sometimes, especially in the cones, visibly intermixed with transparent material. It will be seen, hereafter, that the prevalence of this glassy matter in the convoluted tubes is one of the characteristics of the intertubular disease. This is suggestive of an explanation. As effusion takes place between the tubes; it easily drains through the thin, basement-membrane, and dislodges the epithelium upon it. After a time the cells cease to be reproduced, and the tube remains naked, containing only the transparent, fibrinous portion of the exudation.

It has been supposed that this disorder essentially consists in a degenerative change in the epithelial cells, but my own observations have abundantly convinced me that this is not the case, the cells in the earlier stages of the complaint being usually exactly what they are in a state of health. The epithelial cell loses its character very soon after death. In kidneys, apparently healthy, we always find that a certain proportion of the cells have suffered the loss of their outer coat, so that they are seen as mere nuclei. This is probably a post-mortem change, and it is not more common in granular than in healthy kidneys.

I have alluded to some of the essentials of the disorder in speaking of its earliest stage. I will now give a brief summary of the results of the intertubular disease when it assumes a chronic form.

A very general result of this change is a diminution in size; the kidney often being no more than a third of its original weight. The capsule is thickened and adherent, as in the earliest stage, and manifests a tendency to split into two layers, leaving upon the kidney a very thin film, hard to separate from the renal substance. I have fancied that this may owe its existence, not so much to a splitting of the original capsule, as to an effusion from the surface of the kidney becoming converted into an inner vesture, more or less closely attached to the outer. If there be here an intertubular exudation, there is nothing but the pressure of the capsule to prevent the effusion showing itself superficially. On the removal of the capsule the surface is seen to be uniformly and finely granular, often looking as if each of the little fictitious lobules on the surface had become depressed at the edges. The colour remains pretty much what it was in health, or has more of red mingled with the natural brown. On section the diminished cortical structure presents a coarse texture, which, with its dirty-red tint, often reminds one of sandstone. Cysts, large and small, are frequently observed.

With this state of kidney, I may remark, in passing, that the urine may contain little or no albumen, and that there is usually no dropsy, nor, indeed, any other direct indication of its presence. It often appears as one of the accompaniments of age. I dwell upon it here, chiefly because it supplies a convenient step to the exposition of the more noticeable forms of the granular disease, although it is by no means devoid of clinical interest.

In a magnified section we find that all the central parts of the organ look natural, but near the surface the characteristic changes manifest themselves. Sometimes narrow-pointed processes of fibrous material are seen passing a little way into the kidney from the intergranular depressions. If these alone are seen it may be inferred that the specimen is a very early one. Generally the section shows in the same position the contracted remains of the tubes imbedded in some sort of effused matter. Occasionally these contracted remains are all that indicate the path of the exudation. We may presume that this destruction of tubes is not owing to any disease within themselves, for the change extends quite independently of the direction they take. It usually follows a straight course inwards, irrespective of the bends and tortuosities of the ducts. If the section happens to come across a vessel, it will be seen that it is surrounded by an increased quantity of fibrous tissue. The capillaries are thus obstructed, and the transmitting power of the organ proportionally reduced. (Plate IX, fig. 1.)

With the fresh kidney, and a high power, we may add a few particulars. Some of the tortuous ducts are in a natural condition; mingled with them are others irregularly filled with their own secretion in a comminuted state. This is a very constant condition, and is I believe essential to the disease. Excepting that transparent fibrinous matter is sometimes found in the straight tubes, the state of the cones is much the same with that of the cortical portion. There is to be observed the same predominance of natural structure, and the same irregular occurrence of ducts containing granular matter. The condition of the tubes is in some respects the same as with the smooth kidney. The main point of distinction appears to be this: In the smooth kidney we have an excessive secretion of epithelium which is universal, affecting alike all the secreting structure. With the granular we have an irregular shedding of the epithelium, which is only partial, some ducts being affected while their neighbours escape. This is exactly what we should expect as the consequence of an intertubular effusion taking place at intervals through the organ.

In the chronic granular kidney, cysts are often developed. They are of various sizes, but chiefly of rather greater diameter than the tubes. Occasionally they are as small as the smallest. In texture they exactly resemble the ducts among which they lie. In the cones they are frequently elongated, and placed end to end, where, to compare great things to small, they much resemble a string of sausages. No impartial observer can doubt that in granular kidneys they result from the transformation of tubes. It seems impossible that they should be constructed merely by the blocking up of the ducts. The secretion which fills them is of course incapable of organization. It is necessary, therefore, that before a continuous cell can be produced, their sides must be brought into contact and must grow together. This can only be done by external pressure exerted upon the tubes at intervals, and the gradually contracting matrix of the kidney at once supplies the condition.

I shall now proceed to indicate wherein what I have called the acute granular disease, differs in its results from the chronic. As one variety passes by imperceptible steps into the other, it is not very easy to fix a line of separation; although well-marked cases of each are sufficiently distinct, both in their morbid anatomy and in their effects on the system.

The condition of kidney which I have now to describe, unlike what I have called the chronic intertubular disorder, is always accompanied with albuminous urine, and generally with some degree of dropsy or some other obvious indication of deranged function. It is characterised by an irregular surface, covered with prominent light coloured granules, which project from a purplish ground. Sometimes light buff is the prevailing colour both of the elevations and depressions, the hue of course varying with the degree of congestion. These granulations vary in size, being commonly about one sixteenth of an inch in diameter. They are larger and more distinct than in the other variety of intertubular disease. On section, the cortex generally narrowed, looks as if made up of two materials coarsely intermingled, often reminding one of the texture of fine granite. Cysts are of very general occurrence.

A magnified section of a kidney in this condition is represented in the drawing, No. 5. The morbid appearances existing in the chronic form are found here further advanced. There is an obvious increase of fibrous tissue in the organ. Next to the capsule is often seen a mass of structure, in which all the renal elements have disappeared except the Malpighian bodies, which present more resistance to the compressing agency than the tubes. The ducts which remain are apt to be irregularly dilated. Occasionally may be seen passing into the organ a narrow portion of exudation which at first displays no tubes in its interior, but as it advances becomes spread out so as to include them, and when it is about to cease, its extent may be indicated only by a dark shade over the renal structure.

By the more usual, though less comprehensive, method of examination, we learn that a great characteristic of this form of disease is the peculiar condition of the cortical tubes. When the disease has lasted for any length of time they become devoid of lining, and appear either empty, or filled with glassy matter. This condition must necessarily take some time to become developed; the longer the complaint has lasted the more bare the tubes become. Taking nine well-marked cases, this process of denudation was found to have commenced in all but two. The fibrinous matter in some cases had become thickly sprinkled in its interior with oil-globules, the result of some change within itself. This, of course, is seen mingled with the natural growth in the earlier stages of the disease. The presence of the glassy material in the convoluted tubes, instead of, as in the smooth kidney, their own epithelium, can be conjecturally accounted for. If we suppose a fluid effusion to arise from the capillaries or from the fibrous skeleton of the organ, among and between the tubes, we should expect it to soak through the thin tube-membrane, and undermine and remove many successive crops of epithelium. In the more acute cases, the cells, at length, cease to be reproduced, and the tubes become filled with the fibrinous infiltration.

The ducts which compose the cones exhibit the same changes, but to a less extent. Usually some tubes remain unaffected by the disease, a few may contain disintegrated epithelium, but, as in the cortex, the greater number are empty or filled with translucent fibrinous material.

It is worth observing that in granular kidneys the tubes, of both kinds, are more difficult to isolate than in the smooth. Any one who has been in the habit of scratching the texture into shreds, for microscopic examination, cannot fail to have noticed this.

This sketch of granular disease of the kidney would not be complete without a few words upon the condition of the epithelium, especially as the complaint has been supposed to consist essentially in an atrophy or degeneration of the secreting cells. Much that I have already said tends to show that this is not the case. I carefully examined the epithelium in twenty-seven granular kidneys, taken without selection as they occurred in the dead-house. In five of these the cells contained more or less oil. The quantity was generally small. This is different from what is found in the other forms of renal disease, where the epithelium contains oil in two out of three cases. In five others of the specimens so examined, a certain quantity of oil was found in the tubes, though none could be seen in the separated epithelial cells. This probably had been developed in the fibrinous exudation I have before spoken of.

I have not been able to detect any other change in this portion of the organ. I have carefully compared the epithelium from granular kidneys with that from the same glands in a state of health. In both kinds I have found it to vary much—being sometimes granular, ragged, and ill defined; and sometimes, especially when decomposition has commenced, showing nothing but little sharp-edged bodies, spherical or ovoid in shape, which are the isolated nuclei. I have not been able to find that these conditions are more frequent in diseased than in healthy kidneys, and I believe them to be the result of post-mortem change.

In conclusion, I wish to give briefly the results of some experiments upon circulation in the kidney in health and disease. At the suggestion of Dr. Bence Jones an apparatus was arranged by which water could be poured into the artery of different kidneys under the same circumstances as to pressure, temperature, and size of orifice. The quantity of fluid which then escaped from the vein in a certain time was taken as affording a measure of the permeability of the capillary system in each case.

A cistern containing a definite quantity was so placed as to give a column of water with a fall of eight feet four inches. This was discharged into the kidney which was the subject of the experiment, through a flexible tube terminating in a metal nozzle of such a size as to enter a renal artery of the smallest calibre. The water in the cistern was always heated to 110°, for it was found that then the loss of heat in the tubes was such that the fluid passed through the organ with the least attainable variation from 99°, which was taken as blood heat. The kidneys were all supported in the same position, and they were all operated on at not less than thirty-six hours after death, and before there was any evidence of decomposition. Water was in this way conducted through thirteen healthy kidneys, through five well-marked specimens of the large smooth variety, and through six equally definite examples of granular degeneration. The following table shows the quantity of water which passed through the organ in ten minutes in each case, and gives at the same time the weight of the kidney which was the subject of the experiment:

Table showing discharge from vein in ten minutes.

HEALTHY.		LARGE SMOOTH.		GRANULAR.	
Weight.	Discharge.	Weight.	Discharge.	Weight.	Discharge.
—	53 oz. 1 dr.	11 oz.	129½ oz.	"Shrunk"	40 oz. 5 dr.
5¼ oz.	59	11½	89½	6 oz.	15
7½	220	10½	81	6	22
8	52	7 (æt. 12)	66½	2½	4 6
5¾	84	9¼	86	4	26
4¾	105			4¾	43
5	90				
7½	221				
6½	61				
5¾	72				
7½	145				
6¾	200½				
7½	196½				

From this it appears that, though great variation exists within the limits of health, yet the effect of disease is sufficiently obvious. The average discharge of the healthy kidney proved to be about 119 ounces, the smallest amount being 52 ounces, the largest 221 ounces. The circulating powers of the large smooth kidney do not depart very notably from this—the escape varying from 66½ ounces, which was in the case of a child, to 129½. Thus it appears that the large mottled kidney allows the normal quantity of blood to pass through it, although its transmitting power is not commensurate with its gain in size. This is what might have been expected, considering that the increased weight is merely due to accumulated secretion. The appearance of anæmia results, not from absolute deficiency of blood in the organ, but from the quantity of opaque epithelium by which the blood-vessels are separated.

With the granular kidney the results are more characteristic. The average flow here was little more than 25 ounces; less than a quarter of what it is in health—the kidney where the obstruction was greatest discharging only 4¾. The smallest quantity delivered by the healthy organ was 52 ounces; by the large smooth variety, 66½. With the granular the greatest did not reach either of these amounts, being only 43 ounces. In a specimen not far removed from health, where the granulations appeared to be just commencing, I found the discharge reached 56 ounces. I have not included this in my table, as not being a well-marked case, the condition of the kidney being doubtful.

It is sufficiently manifest that, as a kidney becomes granular, it becomes impervious, and cannot, as a rule, receive more than a small part of the blood which should traverse it. The condition is, to use Dr. Prout's term, one of anæmotrophy. The obstruction to the circulation takes place in the ultimate vessels, and not in the main trunks. I have made careful measurements of the diameter of the vessels in granular kidneys as compared with those in health. I will not prolong this paper by needless details; but I found that the internal circumference of the renal vessels could, in most cases, be accurately gauged at a spot between the final union of all the vessels from the gland, and the attachment to the aorta or vena cava. Measuring at this spot, I found the internal circumference of the artery in health varied, in thirteen cases, from 0·45 to 0·75 inch. The corresponding vein was ascertained in eleven cases. It varied from 1·07 to 1·52 inch. Now, in the granular kidney, which discharged only 4½ ounces, the artery measured 0·45, the vein 0·94. In the specimen which delivered 26 ounces, the artery measured 0·46; the vein, 0·91 inch. And in other instances I have found the measurements almost identical with these. Thus it appears that the blood-vessels are of somewhat diminished capacity, especially the vein, but that the diminution is utterly insufficient to account for the impediment to the circulation. The slight narrowing of the trunk-vessels is probably due to the power they have of adapting themselves to the smaller quantity of blood which is passing through them. The pressure of blood upon the artery, of course, is what it always was, while that upon the vein is diminished as the capillaries of the organ become obstructed.

The chain of anatomical evidence in favour of the intertubular origin of the disease which produces the granular kidney is tolerably complete. It may be thus summed up:

1. Near the surface an effusion is often seen obviously between the tubes. This accounts for the superficial granulation, and bears out the analogy between granular degeneration of the kidney and cirrhosis of the liver.

2. Tubes are caused to shrivel by some influence which travels independently of their direction.

3. Tubes are sometimes divided into cysts. This can only be accomplished by a contractile force external to themselves. In the smooth kidney this never occurs.

4. In most cases some of the secreting tubes are found natural, as if they had been out of the way of the effusion, whereas in the smooth kidney all are commonly involved.

5. A decided difference exists in well-marked cases of each sort in the contents of the tubes. In one they are filled by their own morbid secretion; in the other they are occupied by a material which is foreign to it.

6. In the granular kidney, as in the cirrhosed liver, some alteration takes place in the minute anatomy of the organ which impedes the passage of blood through it. This does not take place in the smooth kidney.

I will not tax the patience of the Society further at present. I hope to be allowed to bring forward some clinical facts in connection with the subject on a future occasion.

DESCRIPTION OF PLATES.

Plate VII. Healthy kidney.

A section of the cortical structure, made in the manner described, showing the arrangement of the tubes and Malpighian bodies. The edge of the section corresponds with the surface of the organ (p. 226).

Plate VIII. Tubular disease.

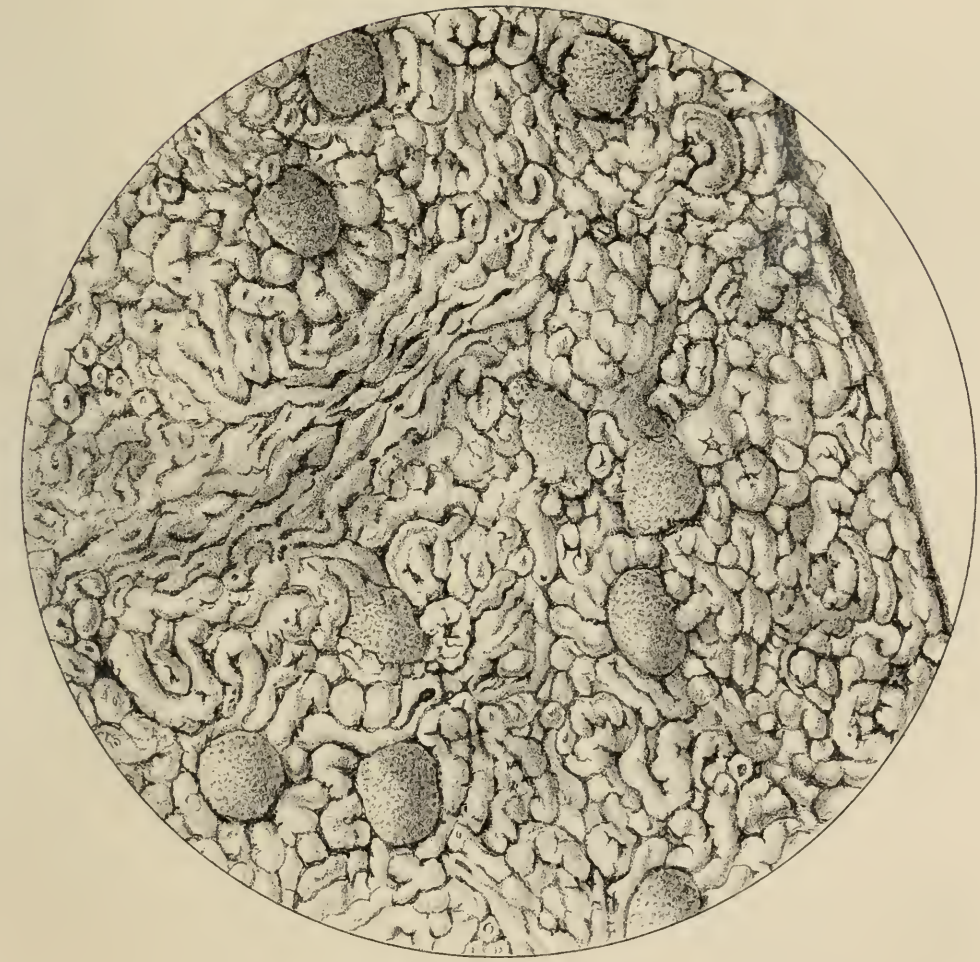
Fig. 1. A section, made in the same way, from a large, smooth kidney, where, however, the process of shrinking had commenced. Some tubes are in a state of distension, while others have burst and collapsed (p. 233).

Fig. 2 shows the condition where the same changes have gone on to a further extent, producing much diminution of bulk. Some tubes are still swollen, but most are shrivelled. The Malpighian bodies are enlarged and irregular in shape; they are closely approximated, owing to the destruction of the tissue between them (p. 234).

Plate IX. Inter-tubular disease.

Fig. 1, showing the exudation in the chronic or partial variety. The inter-tubular effusion, giving a dark colour to the section, is seen commencing near the surface, which has become depressed in consequence. The tubes are compressed where the exudation has taken place; they may be recognised by their lighter colour. Out of its way they remain natural (p. 238).

Fig. 2. A section exhibiting the more extensive changes characterising the severer forms of the disorder. The inter-tubular effusion is well seen, starting at the surface, and becoming diffused as it proceeds inwards. The capsule still remains attached; close to it the processes of exudation may be seen palpably between the tubes (p. 240).



Magnified 75 Diameters.

Natural Condition.

T.West del. G.H.Ford lith.

W.West imp.

Plate VIII.(or N^{os} 2 & 3.)

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Fig 1

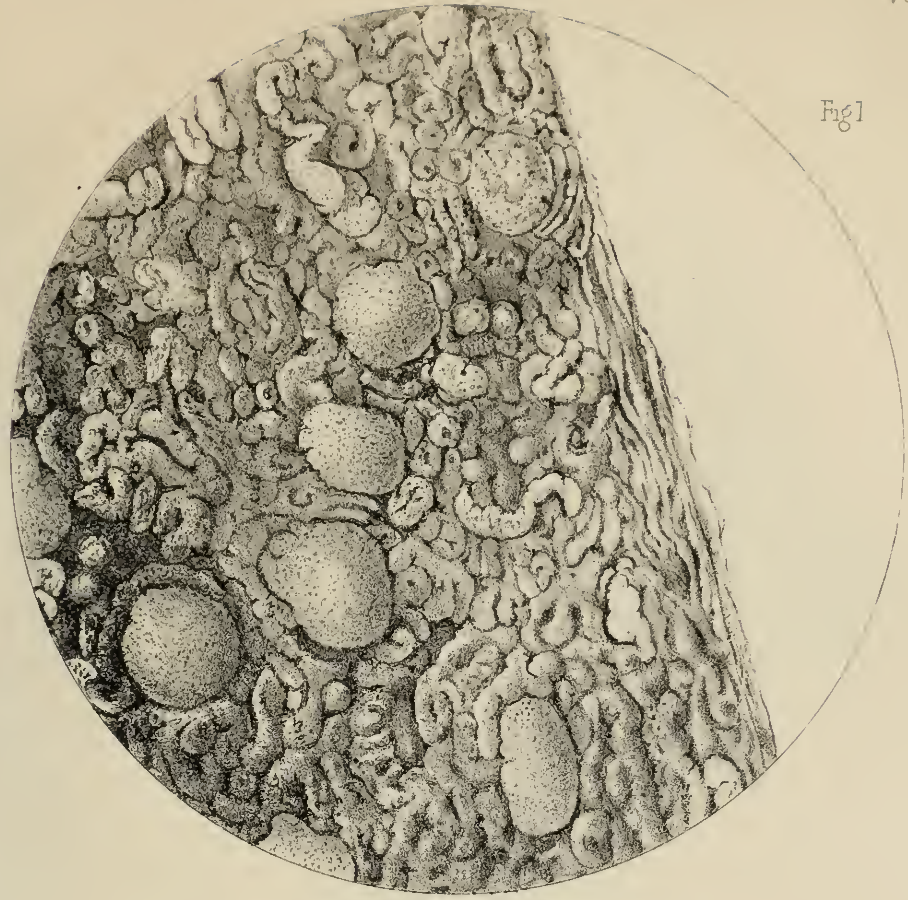
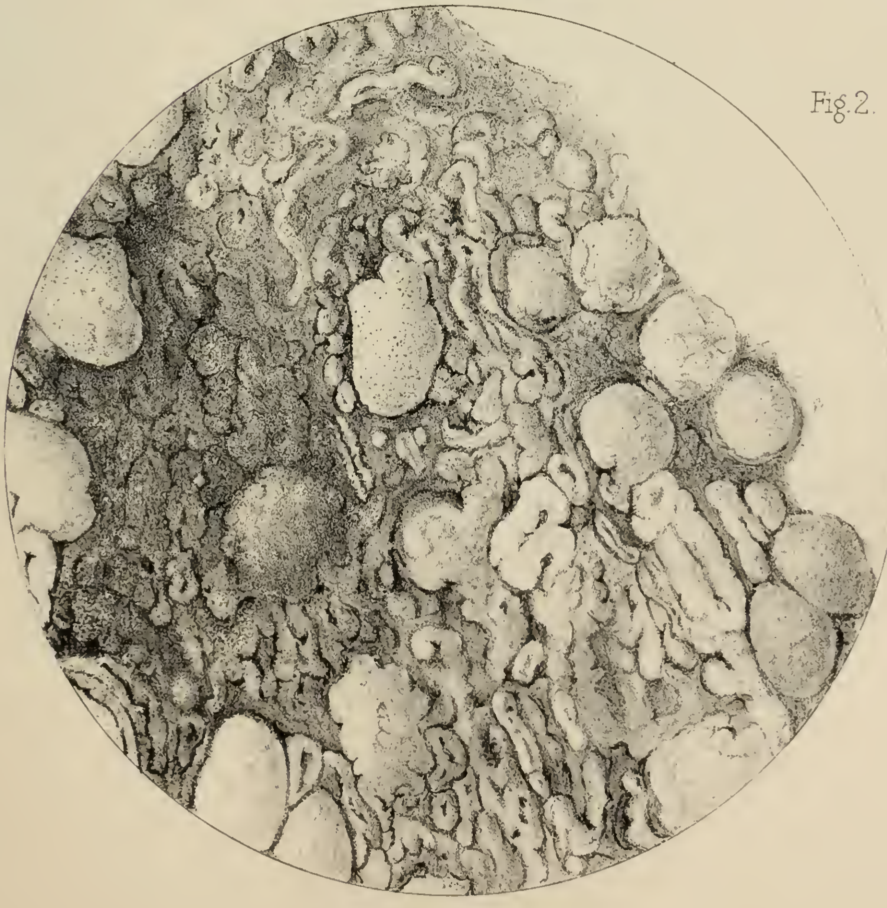


Fig. 2.



Magnified 75 Diameters.

T. West del. G. H. Ford lith.

Tubular Disease

W. West imp.

Fig. 1.

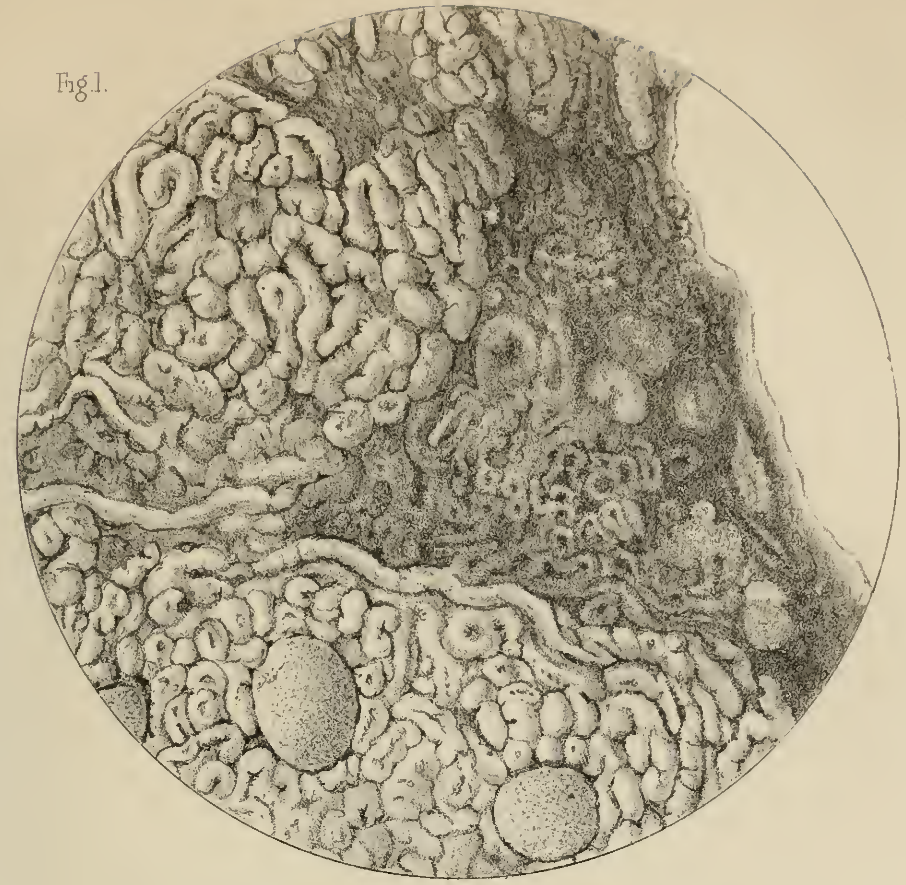
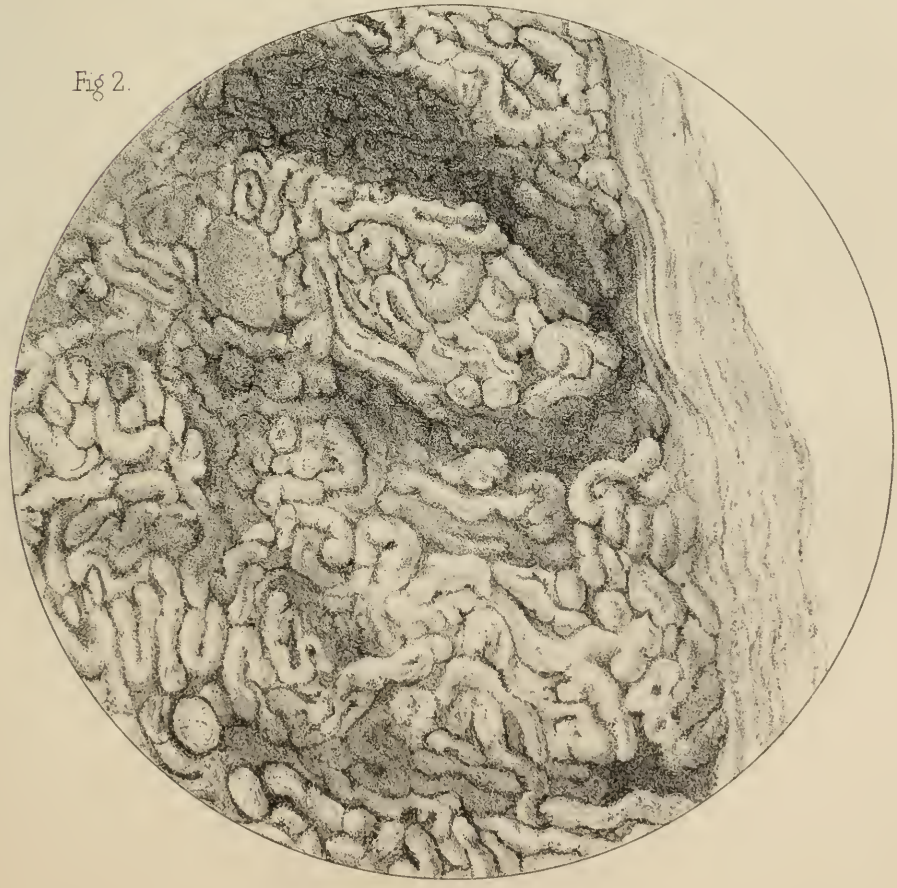


Fig 2.



Magnified 75 Diameters

T West del G.H Ford lith

W West imp

Intertubular Disease

GLAUCOMA,

AND ITS

SURGICAL TREATMENT BY IRIDECTOMY.

BY
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In December, 1857, I laid before this Society an account of some points in the morbid anatomy and pathology of glaucoma, drawn from dissections of eyes made directly after their extirpation, and whilst perfectly fresh. In the discussion which arose upon this communication, mention was made of the treatment of glaucoma by "Iridectomy," an operation first practised by Graefe in chronic irido-choroiditis with posterior synechiæ, and subsequently adopted by him with remarkable success in the treatment of glaucoma. In the following spring, I contributed to the 'Medical Times and Gazette' a short notice of Graefe's operation, which had been tried upon several patients in the Royal London Ophthalmic Hospital, and which already, at that time, promised most satisfactory results. Since then, iridectomy has been very extensively practised in cases of glaucoma by some of my colleagues (of whom I would especially mention Messrs. Bowman and Critchett) and by myself, with a success which has entirely assured us of the great value of the operation. In England generally, however, it has hitherto met with much opposition, and has gained but few advocates beyond those connected with the hospital where it was first practised: it must therefore, in the absence of a general recognition of its beneficial effects in glaucoma, be considered, in this country, as still upon trial.

Being convinced by the accumulated experience of many cases, during the past two years, of the curability of glaucoma by iridectomy, and feeling persuaded that this treatment only requires a fair and impartial trial to secure its general adoption, I am desirous of bringing it under the notice of this Society. Of the several objections which have from time to time appeared in the journals against it, the principal one is its alleged uniform failure, even in the hands of very experienced surgeons. I can only attribute this to its having been applied to cases that were not true examples of glaucoma; an occurrence rendered highly probable by the absence of a general agreement respecting the nature, symptoms, &c., of this disease. On this account, in order to prevent any misunderstanding respecting the nature of the cases in which I advocate the performance of iridectomy, I shall preface my remarks on the operation with an outline of glaucoma, referring those who desire a more detailed account to Graefe's masterly and comprehensive papers in the 'Archiv für Ophthalmologie.'

Glaucoma occurs in two forms, an acute and a chronic, which differ mainly in the relative duration and intensity of their symptoms, whilst their affinity is rendered evident by community of structural changes, by a common termination, and by the fact that chronic glaucoma sometimes passes into acute. There is also a third class of cases occupying an intermediate position.

Prodroma.

In 75 per cent. or more of all the cases, the active stage of the disease is preceded by a premonitory period—"*Prodroma.*" The transition from this to the active period is sudden and marked in acute, whilst it occurs by insensible gradations in chronic glaucoma. As it is a disease almost peculiar to mid-life and old age, its subjects are for the most part presbyopic; but a rapidly increasing presbyopia is always suspicious, and often foreshadows glaucoma. The appearance of a rainbow round the flame of a candle, occasional spontaneous flashes, and other luminous spectra, are early symptoms of common occurrence. At this time, too, an occasional dimness of vision is noticed, often towards the close of day, or under the influence of excitement, and this is associated with vague pain in the eye and about the brow and temple. The pupil is rather large and sluggish; the iris and lens are pushed slightly forwards, and the size of the anterior chamber is diminished. A slight hardness of the globe, indicating increased tension, may generally now be detected by careful palpation. The duration of these premonitory symptoms is very variable; it may be restricted to a few days, or may extend over weeks, months, or even two or three years. Their intensity also varies greatly, and they are frequently so slight that they do not excite serious apprehension; indeed this happens commonly amongst the poor and labouring classes.

Acute Glaucoma.

In acute glaucoma, the prodromal symptoms are generally followed by a sudden attack of violent pain in the eyeball, with rapid extinction of sight, often attended with sickness. The pupil is widely dilated and motionless, and it sometimes has that peculiar greenish tint which was formerly considered so characteristic of glaucoma. The ciliary region and the conjunctiva are red, and the latter is often slightly chemosed. The globe is very hard. The cornea is dull, and its sensibility is diminished. After a variable time, a remission takes place: the eyeball becomes less hard, the pain abates, the redness subsides, and sight returns. This, however, is not a cessation of the disease, and the temporary improvement is sure to be followed by fresh paroxysms, which sooner or later invariably end in complete blindness.

The eyeball remains hard and painful for a long time after sight is extinct, and sclerotic staphylomata frequently form; a very common situation for them being just behind the insertions of the tendons of the musculi recti, where the coat is naturally thinnest and least resistent.

Chronic Glaucoma.

In chronic glaucoma the premonitory gradually slides into the active period. The obscurations and pain, at first evanescent and separated by long and complete intermissions, become more frequent and last longer, until at length they are never wholly absent. The globe becomes harder. The area of the field of vision decreases. The aqueous humour becomes slightly clouded; the pupil is dilated, and sometimes adherent to the capsule of the lens; the iris is dull, and enlarged veinlets may often be seen upon it. The lens, pushed forwards by excessive pressure from behind, closely approaches the cornea, lessening the size of the anterior chamber, which is also encroached on laterally by the iris, distended with fluid which has accumulated in the posterior chamber. The sclerotic assumes a waxy, leaden hue. In the ciliary region, the distended efferent veinlets encircle the cornea with arches, and send backwards varicose trunks. The conjunctiva becomes atrophied, and is readily torn. The cornea is dull, its convexity decreases, its epithelium is raised in minute vesicles, and its sensitiveness is greatly impaired. By this time vision is reduced to a slight quantitative perception of light, or is wholly extinct. At this stage it cannot always be determined from the appearance of the eyeball whether the disease had originally an acute or chronic type, but I have observed that sclerotic staphylomata are more common in the originally acute cases.

I would call particular attention to the flattening of the cornea in glaucoma; because a statement has recently appeared that the cornea acquires increased prominence, becoming, in fact, conical in this disease. Its flattening may be readily demonstrated, as Graefe observes, by comparing the image which the flame of a candle forms upon the cornea of the glaucomatous eye with that formed on the cornea of the sound eye. It has been experimentally shown that excessive distension of the eyeball lessens the curvature of the cornea, and no doubt the flattening in glaucoma is due to this cause.

Ophthalmoscopic Signs.

Excavation of the optic nerve-entrance and pulsation of the arteria centralis retinæ are pathognomonic of glaucoma. Small dotted hæmorrhages in the retina are of frequent occurrence, and are occasionally associated with delicate filmy blood-clots in the vitreous humour. The excavation of the optic nerve-entrance is indicated by a bluish-gray colour of its periphery, and by a peculiar arrangement of the veins. Where the excavation is slight, they undergo a sudden diminution of calibre at the margin, being smaller within the limits of the nerve-entrance than on the adjacent fundus; but where the hollow is deeper, the veins are abruptly bent, or their continuity is apparently interrupted, at the spot just mentioned. The excavation, though partly the direct mechanical effect of the excessive tension of the eyeball, is mainly a secondary consequence of it: the excavation is begun by compression of the soft, yielding nerve-substance, and afterwards increased by its atrophy.

In extreme excavation the bottom of the hollow lies outside the level of the choroidal foramen, and is formed by the lamina cribrosa, covered by the detritus of the wasted nerve-fibres. I have recently dissected an eyeball in which the lamina cribrosa itself was pushed outwards; in this case the excavation was very deep. The bluish colour of the nerve-entrance is the combined effect of shadow, and the peculiar reflection given by the fibrous tissues and fatty granular remains of the nerve. The spontaneous pulsation of the retinal vessels is another effect of excessive intra-ocular pressure. A less degree of pressure suffices to produce the venous than the arterial pulse; and it was shown long ago that both may be artificially produced in the healthy eye by compressing it with the finger.

The retinal hæmorrhages come from ruptured capillaries which have become varicose and sacculated from over-distension. I have already described this condition of the capillaries in a former paper. Very erroneous opinions have prevailed respecting the state of the vitreous humour; by many it was supposed to be fluid—a misconception that has given rise to futile plans of treatment. In acute and in chronic glaucoma, the vitreous humour continues, for a long time, much firmer than natural. In several eyes which I have dissected at an early period of the disease, I have been much struck with the remarkable consistence of this humour, and I have verified the existence of the same condition in the living eye by puncturing the sclerotic freely with an extraction-knife, and then endeavouring to squeeze out some of it. That a *dissolved* state of this humour is an *essential part* of glaucoma, is wholly opposed to my experience. It is only in old cases, where the disease has run its course, and when the retina, the choroid, and all the other tissues of the globe are involved in a common atrophy, that the vitreous humour becomes diffluent; and I believe that those dissections in which it has been found fluid must have been made at this late period. In a recent dissection of acute glaucoma, I found a thin stratum of yellow serum between the hyaloid capsule of the vitreous humour and the membrana limitans of the retina. I had previously suspected the possibility of such an occurrence from ophthalmoscopic observations on the living eye.

The Nature and Causes of the Glaucomatous process.

All the prominent symptoms—the hardness of the globe, the peculiar progressive contraction of the field of vision, the paralysed dilated pupil, the intense throbbing pain, the excavation of the optic nerve-entrance, the pulsation of the retinal vessels—are, one and all, consequences of excessive intra-ocular pressure, and this is due to an increase of the vitreous humour, both as regards quantity and firmness. In a very typical case of acute glaucoma, I found, upon dissection, the hollow of a large sclerotic staphyloma, beneath the tendon of the musculus rectus externus, wholly filled with very firm vitreous humour, in no way differing from the remainder. Since the vitreous humour draws its nourishment from the vascular tunics, any increase of it must depend on some disturbance of the process of nutrition in these parts; and there are good grounds for believing that a congested or inflammatory condition of the choroid is the first step. Graefe considers "acute glaucoma" to be a serous choroiditis, and I am disposed to adopt his view. Although I have not so frequently verified, with the ophthalmoscope, the presence of choroidal hæmorrhages in the living eye in glaucoma as he has done, and have only exceptionally observed them in dissections, I have often ascertained, by actual measurement, that a considerable dilatation of the larger veins exists, and of the choroidal capillaries. Probably the retina is only passively concerned. The calibre of its arteries is scarcely enlarged, the arterial walls become hypertrophied; but this and the venous and capillary hyperæmia seem to be due to obstruction of the efflux of blood at the optic nerve-entrance. The occasional outbreak of glaucomatous inflammation in amaurotic eyes, where the retina has long been atrophied, also shows that this tunic plays no essential part in the disease. I cannot pass without notice an ingenious theory of glaucoma which Mr. Hancock has recently propounded. He attributes the pain, the ophthalmoscopic signs, and the pathological appearances, to a spasmodic contraction of the ciliary muscle (analogous to spasm of the urethral muscles and sphincter ani) compressing the ciliary nerves, and obstructing the circulation. He has observed, in acute glaucoma, a circular depression, externally, corresponding to the ciliary muscle, and an elongation of the antero-posterior diameter of the eyeball, with conicity of the cornea, which he also ascribes to the same muscular spasm.

It is obvious that the cases in which Mr. Hancock saw these features cannot have been examples of glaucoma, such as I have described it; because, as I have stated, the cornea is flattened, and not conical. Allowing, however, that a spasm did exist, the contractility of the muscle would soon become exhausted by the continuous tension to which it is subjected. But the most complete proof that the spasm of the ciliary muscle is not an essential part of glaucoma is afforded by the coexistence of a hard, painful, glaucomatous state of the eyeball, with advanced atrophy, and fatty degeneration of the muscle, a fact which I have several times verified by dissection. Assuming it to be highly probable that the glaucomatous process begins as hyperæmia or inflammation of the choroid, the question next arises, to what is this due? It has long been customary to connect glaucoma with gout, but, as it appears to me, without sufficient evidence. A very large number of cases occur in women, without gouty history or hereditary tendency, and very many of its male subjects do not present any trace of the gouty diathesis. Atheroma of the vascular system has been assigned as another cause; and, inasmuch as glaucoma generally attacks persons of or beyond middle age, an earthy, rigid state of the arteries is often present: but I have so constantly failed to discover any degeneration of the vessels of the eye in typical cases of glaucoma, at an early period, that I am persuaded the two diseases do not stand in the relation of cause and effect. I have endeavoured to ascertain whether kidney disease, with albuminuria or diabetes, have any immediate connection with glaucoma, but with negative results; yet kidney disease and retinal apoplexy are frequently associated. At present we only know, that a certain period of life, an enfeebled constitution, prolonged mental anxiety, and broken rest, predispose to its occurrence. Some injuries and diseases of the eyeball occasionally lead to a glaucomatous condition. Penetrating wounds of the ciliary region, injuring the lens, are prone to do so, as I have seen in several instances. The same fact had been remarked by Graefe, who had also noticed its occasional occurrence in advanced sclerotico-choroidal staphyloma and chronic irido-choroiditis. I have also observed this, and I have verified by dissection the presence of a hollowed optic nerve-entrance in advanced posterior staphyloma.

Prognosis.

Blindness is the natural issue of glaucoma.

Treatment.

The age and broken constitution of the subjects of glaucoma usually forbid general depletion. Venesection is inadmissible; and I have never seen mercury or aconite, though freely administered, cure a single case. Leeching and blistering are occasionally useful auxiliary measures; but alone they are quite incompetent to cope with the disease.

The over-tension of the eyeball naturally suggests the evacuation of some of the superabundant fluid by tapping, an operation with which some of the older surgeons, Antonius Nuck, Riverius, Jobus à Meekren, and others, were familiar; but they appear to have chiefly practised it in onyx, hypopion, and hydrophthalmos. Wardrop, whose observations are contained in the fourth volume of our 'Transactions' (1813), first gave it an extensive trial, and attempted to define the cases in which it was likely to prove useful. With the object of lessening fulness and congestion, he punctured the cornea in superficial, and in deep-seated inflammations of the eyeball. Other surgeons, stimulated by his successes, warmly took up the operation, but the want of uniformity in its results soon led to its being abandoned. In our own day, paracentesis oculi has been warmly advocated by Desmarres in internal inflammation, and some other diseases of the eye; Mr. W. Martin, late Superintendent of the Calcutta Eye Infirmary, also speaks highly of its curative action in fulness of the globe, with pain of a "tensive" character; but amongst English surgeons it has found little favour, though most have occasionally performed it. In some affections the evacuation of the aqueous humour through a puncture in the cornea is a measure of extreme value, and in glaucoma also it affords relief; but this is very transient and incomplete, it does not arrest the course of the disease. Paracentesis of the sclerotic is practised by Desmarres and Hancock, though with different objects, and in different ways; as it has been proposed as a substitute for "Iridectomy," I shall revert to it after considering Graefe's operation.

The limits of this paper do not allow me to notice the various steps by which Graefe arrived at this plan of treatment. The operation consists in excising a segment of the iris, in its whole breadth, from the pupillary edge to its insertion. This is effected through an opening of corresponding size at the extreme margin of the anterior chamber. He introduces the point of a lance-shaped knife through the sclerotic, at about half a line behind the margin of the cornea, and gives it such a direction that it appears in the anterior chamber, just in front of the insertion of the iris, when, by pushing it onwards towards the centre of the chamber, an incision of sufficient size is obtained. Usually, as I understand, he makes an inward iridectomy; but he does not insist on any particular situation; and he says that when desirable, for the sake of appearance, it may be made upwards. The latter situation is that chosen by Mr. Bowman, who first introduced the operation into this country, and has slightly modified some of its details. This modified operation is the one which has been mostly practised in the Royal London Ophthalmic Hospital, and that which I am accustomed to perform. Instead of the lance-shaped knife I have preferred to use an ordinary extraction-knife, as being generally safer. In consequence of the diminished size of the anterior chamber, the incision must be made with care; for if the chamber be opened by a puncture that is afterwards enlarged by a sawing movement, the aqueous humour will probably escape, and the point of the knife may catch the iris, or prick the lens before the incision is completed. The knife, therefore, once inserted, should be pushed steadily onwards across the margin of the chamber, so as to cut its way out at a short distance from the point of entry, ere the escape of the aqueous humour allows the iris to fall on its point or edge. By skirting the margin of the anterior chamber in this manner, the point of the knife is always at a distance from the prominent centre of the lens (which, it must be remembered, closely approaches the cornea in this disease), and there is no danger of wounding it, an accident which I have seen happen several times when the lance-knife has been used. In completing the incision, as the aqueous humour escapes, the iris slightly protrudes through the lips of the wound. It should be gently drawn out with a forceps, and cut off with scissors at each angle of the wound; as this is being done, a drop of yellow serum usually gushes from behind the distended iris. The result is, that the pupil is at once enlarged, up to the incision, which forms, as it were, the base of a coloboma iridis; and the edge of the lens, with the suspensory ligament stretching in front of the vitreous humour, and the ciliary processes, are exposed to view. A little blood oozes into the anterior chamber, from the cut edges of the iris, or from the rupture of enlarged vessels upon its surface. It is well to remove this at once, either by gentle pressure on the cornea, with the smooth back of a scoop, or, if this fail, by passing the instrument into the chamber, through the incision. Should a little delay happen, and the blood have become clotted, it is not readily got away, and may be left to undergo absorption.

The after-treatment is of the simplest kind. Graefe, for a short time after this operation, fastens a light compress upon the eye with an elastic woollen bandage, as a precaution against hæmorrhage. I have sometimes adopted this measure, and have always had reason to be satisfied with it; but I more commonly substitute a piece of wet rag for it. During the next few hours after the operation, the aqueous humour drains away, but the incision soon closes, and the anterior chamber re-fills. The extreme hardness of the eyeball is at once lessened, and a natural tension is gradually assumed. The pain generally at once abates, and soon altogether ceases. As regards vision, my experience is entirely confirmatory of Graefe's, that the ultimate results of iridectomy are intimately dependent on the period at which it has been performed, the restoration being more complete where it has been early undertaken than where it has been postponed.

In the premonitory period, there can be no doubt of the propriety of operating where the intercurrent obscurations, the contraction of the visual field, and the ciliary pain are marked and increasing; and patients will gladly submit to it, if the known undeviating course of the disease be explained to them. I have operated at this period with entire success, arresting the disease, and, as it were, forestalling the impending inflammatory attack. Where the symptoms are slight, and the intermissions long and complete, delay is less dangerous.

In acute glaucoma, I have several times operated at a very early period, during the first paroxysm of violent inflammation, and have obtained most brilliant results. The inflammation has subsided, the eyeball has acquired a natural tension, the humours have cleared, and vision has been very completely restored. I have had patients, who previously to the operation had only a feeble quantitative perception of light, and were wholly unable to recognise the largest objects, who have become again able to read and do needlework.

Some amelioration generally takes place within a few hours or during the next day or two; but the full extent of improvement is not attained till several weeks or months have elapsed. In some instances, the iris regains its brightness and contractility, but it more usually remains motionless. The ophthalmoscope shows a gradual disappearance of the retinal hæmorrhages, an absence of pulsation in the retinal vessels, and, where the operation has been done at an early period before wasting of the nerve, a normal state of the optic-nerve-entrance. In cases such as these, the blindness was due to excessive pressure on the retina, and disappeared on its removal. Graefe predicts complete restoration of vision where moderate quantitative perception of light exists, and the operation is performed before the expiration of a fortnight from the date of the first inflammatory attack. In older cases, the inflammation is subdued by iridectomy, but the improvement of vision is not so complete, its degree depending on the previous progress of the atrophy of the optic nerve and retina. Where the excavation of the nerve-entrance is slight, and where it has not the shining, glistening reflection peculiar to advanced atrophy, it diminishes after iridectomy, as Graefe has shown, and as I have also observed; and I think that it sometimes wholly disappears. The degree of atrophy does not entirely depend on the amount of time which has elapsed since the first outbreak of inflammation, but also on the severity, or comparative mildness of the latter. The less contracted the field of vision, the more favorable the prognosis; yet very successful cases now and then occur, where the area has been previously greatly diminished. In one of my best cases, the day before the acute attack, the visual field, measured at the distance of eighteen inches, was elliptical, the long axis being horizontal; the diameters were nearly three by three-and-a-half inches respectively. In older cases, so long as there is a fair quantitative perception of light, some improvement may be expected. I have realised this, even where I have operated seven and eight months after the date of the first attack.

In chronic, the results of iridectomy are less uniform and less decided, than in acute glaucoma. It is a more insidious disease, and the structural changes creep on simultaneously with the gradual increase of pressure and diminution of sight. Just as happens in the acute form, the earlier the iridectomy, the greater the improvement of vision; but where no improvement takes place, the disease is arrested, and the existing degree of sight is preserved. More rarely, the amelioration is temporary, the globe remains of a natural tension, and the former pain does not recur, but atrophy progresses, and with it sight becomes extinguished. In old cases, where the disease has run its course and sight has long been lost, the pain has generally been relieved; but in a few cases it has either persisted or returned, and excision of the globe has been necessary.

I shall not here indulge in any speculations respecting the manner in which iridectomy reduces the excessive tension of the eyeball, which is the prominent feature of glaucoma: that it does so, is an established fact which, like many others, we may accept empirically until its modus operandi is elucidated by further experience, and a more perfect acquaintance with all the details of the disease.

Alleged objections to Iridectomy.

The alleged uniform failure of iridectomy, in the hands of some surgeons, is, as I have already hinted, with great probability, to be generally attributed to its having been practised in cases which were not examples of glaucoma at all. Other failures have, as I know, proceeded from it having been done in very old cases at far too late a period, when the disease had run its course and the eyeball was disorganized. It has been also said that iridectomy is far too difficult an operation to be generally practised; but its difficulties have been greatly overrated, and I believe that it does not require more skill and manual aptitude than most surgeons possess. When chloroform is used, it really becomes a simple and easy operation; but even supposing it were a difficult one, which it really is not, in the absence of other known means of cure, we should be no more justified in rejecting it on this ground, than we should be in refusing to operate on a strangulated hernia where all other measures had failed. The disfigurement produced by the gap in the iris is too trivial to form a real objection; besides, it may be avoided by forming it under cover of the upper eyelid. The loss of accommodation has been adduced as an objection, but more recent experience has corrected some erroneous impressions which formerly prevailed on this subject.

It is now ascertained that the previously existing presbyopia is not increased by the removal of a portion of the iris; indeed, the refractive power of the globe sometimes actually increases; probably, as Graefe says, in consequence of the flattened cornea resuming a natural convexity.

In order to avoid these alleged disadvantages, paracentesis of the sclerotic has been advocated by Mr. Middlemore and M. Desmarres, as a substitute for iridectomy. The former proposes to evacuate the *turbid diffluent* vitreous humour with a grooved needle, and to replace it with a syringe full of clear water. But the vitreous humour is much too firm to flow out along the groove of a needle, and few English surgeons would, I imagine, adopt Desmarres' plan of introducing a probe and stirring it up. Mr. Hancock, assuming a spasm of the ciliary muscle to be the essence of glaucoma, divides this structure by passing a knife through the junction of the cornea, and the sclerotic and ciliary region, backwards and inwards towards the axis of the globe. In doing this, the margin of the anterior chamber is opened, the aqueous humour escapes, and the vitreous humour is tapped. Having demonstrated that the ciliary muscle is not concerned in maintaining the glaucomatous condition, I am inclined to ascribe Mr. Hancock's reported success solely to the paracentesis: if this surmise be correct, his operation cannot rank as a substitute for iridectomy, until it has been thoroughly established that it permanently relieves excessive intra-ocular tension, which I have found paracentesis of the vitreous humour fail to accomplish.

CONTRIBUTION

TO THE

STUDY OF SPIROMETRY.

BY

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ONE OF THE VICE-PRESIDENTS OF THE SOCIETY.

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A PAPER "On the Capacity of the Lungs, and on the Respiratory Functions," was read before this Society, in 1846, by Mr. John Hutchinson, in which he gave the results of an extensive series of observations, made with great care, by means of instruments invented by himself which ensured a degree of accuracy in measurement not attained previously. The results were highly interesting, and the paper, which was published in the twenty-ninth volume of the 'Transactions' of the Society, is entitled to rank as a valuable addition to our knowledge on this branch of physiological science. Further observation and experiment, however, appeared necessary to determine the utility of the spirometer for some of those practical purposes to which Mr. Hutchinson deemed it applicable.

Shortly after the publication of the paper, the medical officers of the Grenadier Guards procured one of his spirometers, with a view to test its value in the examination of recruits, and to ascertain, if possible, whether any relation exists between the capacity of the lungs, as measured by it, and the tendency to pulmonary disease, as shown in the subsequent medical history of the soldier. For this purpose the height and "vital capacity," as it was termed by Mr. Hutchinson—or the number of cubic inches given by a full expiration following the deepest inspiration—of all the recruits examined at the head-quarters of the regiment were carefully measured and recorded in the recruit-book. Through the kindness of my friend, Mr. George Brown, late Surgeon-Major of the Grenadier Guards, I had access to all the hospital books and records, and was enabled to tabulate the results of these measurements, and to trace the medical history of each recruit enlisted between October, 1848, and March, 1853, from the period of his joining the regiment up to the date of the embarkation of the Guards for the East, in March, 1854. The disturbing influences which then came into operation, were so great as to prevent any accurate deduction being drawn from the subsequent history of the men.

The number of recruits taken on the strength of the regiment during this time, exclusive of a few boys and transfers from other corps, was 1126; the time which elapsed between the date of enlistment of the first man on the list, and that to which the medical histories were traced, was six years and a half; and between the examination of the last man and the same date, was twelve months; taking the average service of the whole of the men, it amounted to three years and five months.

It would have been desirable to have had a larger number of men under observation, and for a longer period; but as many years must elapse before such data can be obtained, I venture to submit this paper as a contribution to the study of spirometry, claiming for it no further credit than that the observations have been accurately recorded, the subsequent history of the men carefully traced, and the results honestly and impartially brought out.

Before entering upon the question of the tendency to pulmonary disease, it may be necessary to examine how far the general results as to "vital capacity" obtained by the medical officers of the Guards, correspond with those of Mr.

Hutchinson. This is accordingly shown in the following table:

Height.	Guards.		Hutchinson.		
	Number of observations.	Mean vital capacity.	Number of observations.	Mean vital capacity.	Capacity calculated in arithmetical progression.
5 feet 7 to 5 feet 8...	17	225·53	411	228	230
5 „ 8 to 5 „ 9...	315	237·51	329	237	238
5 „ 9 to 5 „ 10...	400	242·04	201	246	246
5 „ 10 to 5 „ 11...	235	249·16	116	247	254
5 „ 11 to 6 feet......	113	253·75	112	259	262
6 feet and upwards ...	46	264·00	80	276	270

The vital capacity of men six feet and upwards, as shown in the two series of observations, differs widely; but the comparison is wanting in accuracy, as there is no evidence to show that the proportion greatly exceeding six feet is nearly the same in each series. Indeed, Mr. Hutchinson incidentally mentions two cases in which the height exceeded six feet eleven inches, while none of the Guards at all approached that height, and two such cases in so small a number as eighty observations would of themselves materially affect the results. This opinion is corroborated by the fact that sixty-two observations made by Mr. Hutchinson on seamen, Horse and Foot Guards, and police, gave an average of only 260. As regards the men under six feet, there is a source of difference in the two series, arising from the mode of tabulating the observations; but the extent to which it affects the results can be accurately determined. In Mr. Hutchinson's tables the height has been taken as up to and including the complete inch; while in those of the Guards it has been taken, according to the more usual practice, from the inch inclusive. By the former the height from five feet eight inches to five feet nine inches, does not include the men of five feet eight inches, but those of five feet nine inches; whereas, in the other tables, it comprises men of five feet eight inches, and up to, but not inclusive of, five feet nine inches. As the increment of height is usually taken in eighths of an inch; and as, according to Mr. Hutchinson's observations, the "vital capacity" increases eight cubic inches with every inch of height; this difference in the mode of making the observation would give rise to a difference of one cubic inch of capacity at each height.

It must be obvious that the "vital capacity" stated in the preceding table is the average of men of five feet seven and a half, eight and a half, nine and a half inches, &c.; but if the observations for every two inches of height be combined, and the mean taken, the result will be the average for the exact height of five feet eight, nine, and ten inches, &c. This has been accordingly done in the following table; and at the same time the correction above noted has been made in Mr. Hutchinson's observations.

Average of men of	5 feet 8.	5 feet 9.	5 feet 10.	5 feet 11.	6 feet.
Guards . .	231·5	239·8	245·6	251·5	258·9
Hutchinson . .	231·5	240·5	245·5	252·0	258·5

The identity of the results in the two series of observations, after the necessary corrections have been made to bring them into strict comparison, is very remarkable, and may fairly be deemed an evidence of their accuracy.

Having thus shown that the men under observation had a fair average "vital capacity," we shall proceed to examine the question how far a capacity under the average may be taken as an indication, either of a tendency to pulmonary disease, or of a feeble constitution rendering the man liable to a higher rate of mortality than that to which men of, or above, the average are subject. With a view to test this, the men were divided into three classes:—

1st. Those whose "vital capacity" was more than ten inches under the average calculated for men of the same height by Mr. Hutchinson's tables.

2d. Those whose capacity ranged between ten cubic inches under, and ten above, the average.

3d. Those whose "vital capacity" exceeded the average by more than ten inches.

In the first class there were 416, in the second 342, and in the third 368 men. The average service of the first class, up to the 31st of March, 1854, was 3·632 years, of the second, 3·348, and of the third, 3·277. From these data we find the strength of the first class to have been equal to 1511, of the second to 1145, and of third to 1206 men, under observation for one complete year. The following table shows the number of deaths which occurred in each of these classes.

	Under the average.	Average.	Above the average.	Total.
Strength . . .	1511	1145	1206	3862
Deaths	31	23	24	78
Ratio of deaths per 1000 of strength . . .	20·52	20·09	19·90	20·20

This table shows a remarkable coincidence in the mortality of the three classes, the difference amounting only to 0·6 per 1000 of the strength, in favour of the highest "vital capacity."

But, in addition to the loss to the service by death, forty men were discharged as invalids, and they belonged to the three classes as follows: first class, twenty-seven; second, seven; and third class, six; so that the number who became non-effective was much greater in the men having a "vital capacity" below the average than in the other two classes, though the deaths so nearly approximated.

It was supposed that the spirometer would be valuable in the selection of recruits, by indicating those men in whom a tendency existed to pulmonary disease. In testing the accuracy of this opinion, it must be borne in mind that consumption is one of those diseases in which, from their slow progress, it is easy, and with a view to military efficiency, even necessary to remove the men from the ranks by invaliding. The proportion so discharged, therefore, must be taken into account along with the deaths in estimating the amount of these diseases in each class of Recruits. The results are shown in the following table:

	Aggregate Strength.	By Consumption.			Ratio per 1000 of strength.		
		Died.	Invalided.	Total.	Died.	Invalided.	Total.
Below the average	1511	19	17	26	12·57	11·25	23·82
Average	1145	14	3	17	12·23	2·62	14·85
Above the average	1206	17	3	20	14·09	2·49	16·58

This table shows the loss to the service, by consumption, to have been much greater among the men having a "*vital capacity*" below the average than in the other two classes. The proportion of deaths does not differ materially in the three classes, but the invaliding has been four times as high among the men under the average as among the others. The deaths among the men above the average are rather higher than among those of the average class, but the difference is so small as probably to be attributable to irregularity arising from limited data.

These results appear scarcely to justify the conclusion that a definitive relation exists between the "vital capacity" and the tendency to pulmonary consumption. Had this been the case, the proportion attacked should have been found to decrease among the men above the average.

The legitimate deduction from them seems to be that a "vital capacity" below the average may be considered as indicating a generally feeble organization, less capable of resisting the deteriorating influences to which a soldier is exposed. This conclusion would justify Mr. Hutchinson's opinion that the spirometer might be advantageously employed in judging of the fitness of recruits for the army.

But unfortunately there is a practical difficulty in its application which appears to be insurmountable. If all men under a given "vital capacity" were to be refused, it would be easy for an unwilling recruit to ensure his rejection, as no means exists of testing whether he really inspires and expires to the full extent of his power. The spirometer, however, might be useful as an indication to the Inspecting Officer of the necessity for a careful examination by the stethoscope, to ascertain the actual condition of the lungs and heart, where a very low capacity was shown.

The views expressed by Mr. Hutchinson, of the practical value of his instrument to the Medical Referees of Life Assurance Companies appear to be confirmed by these observations. As there is no inducement for the persons wishing to effect an assurance to mislead, at least in the direction of want of capacity, medical referees would be extremely cautious in recommending the acceptance of any person whose vital capacity was much below the average; and would probably advise the proposal to be declined, if there were any flaw in the personal or family history, and unless the results of a careful examination of the chest by percussion and auscultation were in the highest degree satisfactory.

INQUIRY INTO THE TREATMENT

OF

CONGENITAL IMPERFECTIONS OF THE RECTUM BY OPERATION,

FOUNDED ON AN ANALYSIS OF ONE HUNDRED CASES, NINE OF WHICH OCCURRED IN THE PRACTICE OF THE AUTHOR.

BY

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Of the many congenital imperfections and deficiencies to which the human body is liable, there are few more fatal to the life of the infant, or more distressing to the parents, than those of the terminal portion of the alimentary canal. They are not only varied in form, but the opportunities of observing them occur so rarely, that I fear life is sometimes sacrificed for want of the information necessary to guide the practitioner in dealing with them. In a large number of instances, the operations performed to remedy the imperfection have failed in procuring a vent for the fæces. In many others, where a passage has been made, the relief, though immediate, has not been sufficient or permanent, but has only prolonged a miserable existence; whilst in a very few only has the success been complete, and life been preserved, without risk and serious inconvenience. Indeed, so unsatisfactory have been the results of operative treatment, that some surgeons have objected to interfere at all, and have left the infant to perish. With the view of ascertaining and estimating the results of the operations which have been resorted to in the different forms of these imperfections, either for the preservation of life or its future comfort, and of assisting to establish the best modes of attaining these objects, I have collected and tabulated one hundred cases, in which operations have been performed by myself and other surgeons.

Table of 100 *Cases of Congenital Imperfections of the Rectum subjected to Operation.*[1]

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
1	M.	The author	Imperforate anus	Deep incision and puncture, but rectum not reached	Fatal	—
2	F.	The author	Anus opening into a cul-de-sac; rectum ending in a blind extremity close to the latter	Deep puncture; and gut reached	Fatal, nine days	Rectum reached, but no meconium escaped.
3	M.	The author	Anus opening into a cul-de-sac three quarters of an inch in depth	Incision towards the coccyx, gut reached, drawn down and secured to skin at the anus	Successful, six months	No tendency to contraction, but occasional dilatation continued as a precautionary measure.
4	F.	The author	Imperforate anus; rectum opening into the vagina three quarters of an inch from its orifice	Incision at the anus, and gut reached	Unsuccessful	Great tendency to contract; child neglected, and opening allowed to close after some months. Died from infantile disease at the age of eighteen months.

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[1] In many of the cases in the Table I have been able to add fuller and more recent information than will be found in the references, chiefly from inquiries made of the operators, to whom I beg to express my obligations for their ready assistance. It must be observed that the fatality after operation is much greater than the table indicates, as cases of success are far more frequently put on record than cases of failure or death. Cases of abnormal contraction of the anus, and of simple closure of the aperture by a membrane, without deficiency of the rectum, are not included, the treatment being obvious, and the result generally satisfactory.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
5	F.	The author	Imperforate anus; rectum opening in the perineum, close to the vagina, by a narrow outlet	Incision at the anus, and gut reached	Successful, two years	Operated on at the age of three months; child alive and well at two and a half years of age.
6	F.	The author	Anus opening into a cul-de-sac; rectum wanting; the colon terminating in a blind pouch in the left iliac fossa	Deep puncture, but gut not reached; colotomy in the left loin	Fatal, eighteen hours after second operation	An instrument introduced at the anus had penetrated the vagina, passed behind the uterus, and wounded the peritoneum.
7	F.	The author	Anus opening into a cul-de-sac half an inch in depth.	Deep puncture without reaching the bowel; unsuccessful attempt to open colon in left loin	Fatal, nine days after birth	Descending colon contracted, attached by a mesocolon, and loose in the cavity.
8	F.	Wormald, notes of the author	Imperforate anus; rectum opening into the vagina	Incision and gut reached	Successful, five months	Child brought to the author at the age of five months, having been operated on at St. Bartholomew's Hospital. No attempt had been made to close the opening into the vagina.
9	F.	Adams, assisted by the author	Imperforate anus; rectum opening into the vagina	Incision at the anus, and gut reached	Successful	Operated on at four years of age, the artificial anus easily maintained to the period of the child's death from diphtheria, a month after the operation.
10	M.	Duret, quoted by Amussat in his memoir 'Sur la possibilité d'établir un anus artificiel'	Imperforate anus	Incision, but gut not reached; colotomy in the groin	Successful	Reported by Rochard ('Mémoires de l'Acad. Imp. de Médecine,' tom. xxiii) to have died at forty-three years of age, of a disease unconnected with his infirmity.

11	M.	Desault, ibid.	Imperforate anus; rectum opening into the bladder	Colotomy in the groin	Fatal, four days	——
12	M.	Dumas, ibid.	Imperforate anus; rectum opening into the urethra	Incision and puncture, but gut not reached	Fatal, two days	——
13	M.	Duret, ibid.	Imperforate anus; rectum terminating in a cul-de-sac an inch from the anus	Incision, but gut not reached; colotomy in the groin	Fatal, four days	——
14	F.	Serrand, ibid.	Anus opening into a cul-de-sac an inch and a half in depth	Colotomy in the groin	Successful	Reported by Rochard to be alive and well at forty-six years of age.
15	F.	Miriel, ibid.	Anus opening into a cul-de-sac six lines in depth	Colotomy in the groin	Successful	Reported by Rochard to be alive and well at forty-three years of age.
16	M.	Miriel, ibid.	Anus opening into a cul-de-sac one and a quarter inch in depth	Colotomy in the groin	Successful	Alive at thirteen years of age.
17	M.	Miriel, ibid.	Imperforate anus; rectum terminating in a cul-de-sac some distance from the anus	Incision and puncture, but gut not reached; colotomy in the groin	Successful	Lived twenty-seven months, and died from dentition.
18	M.	Bizet, ibid.	Imperforate anus; rectum wanting	Incision, but gut not reached; colotomy in the groin	Successful	Lived one month. Reported to have died of an indigestion caused by the imprudence of the parents.
19	M.	Klewig, ibid.	Imperforate anus	Incision and puncture, but gut not reached; colotomy in the groin	Successful	Survived three years.
20	F.	Amussat, 'Troisième Mémoire'	Anus opening into the vagina, but not communicating with the rectum, which was entirely deficient; the colon terminating in a blind pouch two inches from the anus	Deep incision behind the anus,. cul-de-sac of bowel reached and drawn into the perineum, and artificial anus established behind the natural anus	Successful	Opening enlarged by Sir P. Crampton, at end of two months; in good health, with power of retention, at nine years of age. Engaged to be married at twenty-one years of age.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
21	M.	Amussat, 'Troisième Mémoire'	Imperforate anus; rectum opening into the bladder between the ureters	Incision in the perineum behind the anus, but gut not reached	Fatal, two days	——
22	M.	Amussat, ibid.	Imperforate anus; rectum terminating in a cul-de-sac an inch from the anus	Incision, and gut reached	Fatal, two days	——
23	F.	Amussat, ibid.	Imperforate anus; rectum terminating in a cul-de-sac	Incision behind the anus, and gut reached and drawn down	Fatal, four days	Death attributed to a prolonged operation.
24	M.	Amussat, ibid.	Anus opening into a cul-de-sac nearly an inch in depth; the rectum terminating in a blind pouch descending behind it	Deep incision behind the anus, and gut reached; septum divided, and communication with natural anus established	Successful	Alive and well at the end of eight weeks.
25	M.	Amussat, ibid.	Anus opening into a cul-de-sac upwards of an inch in depth; rectum wanting	Puncture, but gut not reached; colotomy in the left loin	Successful	Reported by Dr. Alphonse Amussat to have lived to the age of seven years; great difficulty at first in keeping artificial anus patent.
26	M.	Amussat, ibid.	Anus opening into a cul-de-sac upwards of an inch in depth; rectum terminating in a cul-de-sac near the septum	Colotomy in the left loin	Fatal, eight days	——
27	M.	Amussat, ibid.	Imperforate anus	Incision, but gut not reached; colotomy in the left loin	Successful	Alive and well seven weeks afterwards, but known not to have long survived.

28	M.	Bougon, quoted by Amussat	Imperforate anus; rectum terminating in a cul-de-sac an inch from the anus	Incision, but gut not reached; colotomy in the left loin	Fatal, one day	Operations not performed until thirteenth day after birth. Peritoneum opened in colotomy.
29	F.	Sharpless, 'Lancet,' vols. iii and iv	Imperforate anus; rectum terminating in the vagina	Puncture and incision, and gut reached	Successful, four months	Operated on ten days after birth; recto-vaginal opening closed spontaneously in two months after the operation. Dilatation continued at date of success.
30	M.	Edwards, 'Lancet,' vol. i, 1829-30	Anus opening into a cul-de-sac one inch in depth; rectum terminating in a blind pouch	Puncture, but gut not reached	Fatal, twenty-four hours	Died of peritonitis; peritoneum wounded in the operation.
31	F.	Thomas, 'Lancet,' vol. ii, 1830-1	Anus opening into a cul-de-sac; rectum terminating in a blind pouch	Puncture, but gut not reached	Fatal, seven days	——
32	M.	Lindsay, 'Lancet,' vol. i, 1835-6	Imperforate anus	Incision, and gut reached	Successful, six years	The artificial anus originally made at the side of the coccyx was imperfect, and became obstructed with a plumstone at six years of age, and life was endangered. Another opening was made with success and power of retention.
33	M.	Tatham, 'Lancet,' vol. i, 1835-6	Imperforate anus; rectum wanting; colon opening by a narrow canal into the urethra	Incision, and gut reached	Fatal, two months	Unsuccessful in maintaining the opening; meconium continued to pass by the urethra.
34	M.	Smyth, 'Lancet,' vol. i, 1846	Imperforate anus; rectum wanting; colon terminating in a blind pouch	Incision: gut reached, and drawn down the day after the incision, but not secured to the skin	Successful, eighteen months	The incision to reach the gut is reported to have been as deep as the brim of the pelvis.
35	F.	Pickop, 'Lancet,' vol. i, 1850	Anus opening into a cul-de-sac one and a half inch in depth	Incision, and gut reached	Successful, two weeks	——

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
36	M.	Erichsen, 'Lancet,' vol. i, 1851	Imperforate anus; rectum wanting; colon terminating in a blind pouch	Incision and puncture, and gut reached	Fatal, four days	—
37	M.	Clark, Le Gros, Lancet,' vol. i, 1851	Anus opening into a cul-de-sac half an inch in depth; rectum wanting	Incision, and gut reached	Successful, nine years	Difficulty in maintaining the opening; constant attacks of constipation. Second operation performed after three weeks, and a third at a later period. Alive and well at nine years of age, but subject to great inconvenience from feculent accumulations.
38	M.	Arnott, 'Lancet,' vol. i, 1855	Imperforate anus; rectum communicating with urethra	Incision, and gut reached	Successful, nine years	Difficulty in maintaining the opening; incision repeated at end of four months. Alive and well, with power of retention, at nine years of age.
39	M.	Pugh, 'Lancet,' vol. i, 1855	Imperforate anus; rectum opening into urethra or bladder	Incision, and gut reached, drawn down, and secured to skin	Successful, five years	Followed by retraction, instruments required for some time to counteract tendency to close. Alive and well at five years of age.
40	M.	Miller, 'Lancet,' vol. i, 1857, and 'Edinb. Med. Journal,' vol. ii, 1856-7	Imperforate anus; rectum opening into urethra or bladder	Incision and puncture, and gut reached	Successful, thirty-six years	Difficulty in maintaining the opening; repunctured ten times in first eight months. Large alvo-urinary calculus extracted from the rectum at seven years of age. Alive and well at thirty-six years of age; power of retention complete.

41	M.	Dickinson, 'Lancet,' May, 1859	Imperforate anus; rectum opening into the urethra	Incision and puncture, and gut reached	Successful	Operated on at the age of four months, all the fæces passing per urethram previously; operation had to be repeated owing to closure of the wound. Died of bronchitis some months afterwards.
42	M.	Williamson, 'Lond. Med. Gazette,' vol. xxxvii	Imperforate anus; rectum terminating in the prostatic portion of the urethra	Incision, but gut not reached	Fatal, eight months twenty-two days	Fæces passed by the urethra, but obstruction frequently occurred; consent refused to a second operation.
43	M.	Fergusson, 'Practical Surgery'	Imperforate anus; rectum opening into membranous portion of the urethra	Incision, and gut reached	Successful	Died at six years of age from disease of the lungs. No incontinency. Inconvenience continued from communication between the two passages remaining unclosed.
44	M.	Hutchinson, 'Observations in Surgery'	Imperforate anus; rectum opening into membranous portion of the urethra	Incision and puncture, and gut reached	Fatal, ten months	Success only temporary; child died ten months after the operation; the narrow artificial passage, nearly three inches in length, being insufficient.
45	F.	Hutchinson, 'Lond. Med. Gazette,' vol. i	Imperforate anus; rectum terminating in a blind pouch	Puncture, and gut reached	Fatal, five weeks	——
46	M.	Parker, 'New York Journal of Medicine,' vol. xiii, 1855	Anus opening into a cul-de-sac half an inch in depth	Puncture, and gut reached	Fatal, twenty-four hours	——
47	M.	Parker, ibid.	Anus opening into a cul-de-sac	Puncture, and gut reached	Successful	Died at two years of age from croup.
48	F.	Hunter, 'Hunterian Catalogue,' Part V	Anus opening into a cul-de-sac; rectum terminating in a blind pouch	Puncture, and gut reached	Fatal, two days	The barrier was only one twelfth of an inch in thickness.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
49	F.	Dyce, 'Lond. Med. Gazette,' vol. xv	Anus opening into a cul-de-sac one and a half inch in depth	Puncture, and gut reached	Successful, four weeks	—
50	M.	Gorham, 'Lond. Med. Gazette,' vol. xxi	Imperforate anus; rectum terminating in a pouch, but communicating by a narrow canal with the urethra	Incision, but gut not reached	Fatal, a few hours	—
51	M.	Gosse, 'Lond. Med. Gazette,' vol. xli	Imperforate anus	Incision and puncture, and gut reached	Fatal, fourteen days	—
52	M.	Wayte, 'Edin. Med. and Surg. Journal,' vol. xvii	Anus opening into a cul-de-sac an inch deep	Puncture, and gut reached	Fatal, six months	Unsuccessful in maintaining the opening.
53	M.	Redfern Davies, 'Edin. Med. Jour.,' March, 1858	Imperforate anus; rectum terminating in a blind pouch	Incision, and gut reached	Fatal, three days	—
54	M.	Crampton, 'Dub. Med. Press,' vol. xiii, p. 146	Anus opening into a cul-de-sac one and a half inch in depth; rectum terminating in a blind pouch, only a membranous septum intervening.	Puncture, and gut reached	Successful	Dilatation discontinued after six months. Patient alive and well at four years of age, only a slight contraction at the seat of the septum. The functions of the gut quite unimpaired.
55	M.	York, 'Boston Med. and Surg. Journal,' vol. xlii	Imperforate anus; rectum opening into the urethra	Puncture, and gut reached	Unsuccessful	Lived thirteen months, died from a fall. Anus closed, and fæces passed afterwards by the urethra.
56	F.	Higginson, ibid.	Imperforate anus	Puncture and incision, but gut not reached	Fatal, eighteen days	—

57	M.	Jones, ibid.	Anus opening into a cul-de-sac one and a half inch in depth; rectum terminating in a pouch; the septum being thin	Incision, and gut reached	Successful, two and a half years	Bougies discontinued after seven weeks.
58	M.	Gay, ibid.	Imperforate anus; rectum communicating with the urethra or bladder	Incision, but gut not reached	Fatal, two and a half days	——
59	M.	Buckingham, ibid.	Imperforate anus; rectum communicating with the urethra	Incision and puncture, and gut reached	Successful, six years	Great tendency to close for several months, and fæces passed by the urethra. Alive and well six years after the operation, but subject to pain in the bladder and difficult micturition.
60	F.	Corse, 'Amer. Jour. of Med. Sciences,' vol. xxxvii	Anus opening into a cul-de-sac half an inch in depth	Incision, but gut not reached	Fatal, four days	——
61	M.	Chevers, 'Ind. Ann. of Medicine,' vol. i	Imperforate anus; rectum terminating in the urethra or bladder	Incision and puncture, and gut reached	Fatal, three days	Failure in maintaining the opening.
62	M.	Chevers, ibid.	Imperforate anus; rectum terminating in a blind pouch with a narrow canal communicating with the urethra	Incision and puncture, and gut reached	Fatal, thirteen days	Died of peritonitis.
63	F.	Green, 'Ind. Ann. of Med. Science,' vol. v	Imperforate anus; rectum opening into the vagina	Incision at the anus, and gut reached	Unsuccessful	Infant twice operated on, once in 1850, by Dr. Wise, and again in 1852, by Mr. Green. Neglected by its parents. Died in 1854, fæces passing per vaginam.
64	M.	Green, ibid.	Imperforate anus	Incision, but gut not reached; colotomy in groin	Successful	Child well and healthy at age of seventeen months.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
65	M.	Green, 'Ind. Ann. of Med. Science,' vol. v	Imperforate anus; rectum opening into the urethra	Incision, but gut not reached	Fatal, few hours	—
66	M.	Green, ibid.	Imperforate anus; rectum terminating in the perineum just behind the scrotum	Incision, and gut reached	Uncertain	Child not operated on until age of two years. Final result not ascertained; only seen for six days.
67	F.	Green, ibid.	Imperforate anus; rectum terminating in a cul-de-sac	Incision and puncture, and gut reached	Fatal, seven days	The opening made was too small.
68	F.	Green, ibid.	Imperforate anus; rectum opening into the vagina	Incision, and gut reached	Successful	Operated on at age of six months. Doing well at sixteenth day after.
69	F.	Green, ibid.	Imperforate anus; rectum opening into the vagina	Incision, and gut reached	Successful	Operated on at five years of age. Doing well at the end of five weeks.
70	M.	Roux de Brignoles, 'Gaz. Méd.,' 1834	Imperforate anus; rectum communicating with the urethra	Incision, and gut reached	Successful	Difficulty at first in maintaining the passage. Died of scarlatina at the age of three years.
71	M.	Baraduc, 'Gaz. des Hôp.,' quoted in 'Med. Times,' vol. xii	Imperforate anus	Incision, and gut reached	Successful, six years	Rectum nearly an inch from the anus; difficulty in maintaining the passage, and operation repeated twice. Power of retention wanting for three years, and then gained imperfectly. Alive and well at six years of age.
72	M.	Lenoir, recorded by Godard, 'Gaz. Méd. de Paris,' 1855	Imperforate anus; rectum terminating in the urethra	Incision and puncture, but gut not reached; colotomy in the groin	Fatal, ten days	Died from peritonitis and abscess in the pelvis, consequent on the puncture. No meconium escaped with the urine.

73	M.	Guersant, recorded by Godard, ibid.	Imperforate anus; rectum terminating in the prostatic portion of the urethra	Incision and puncture, but gut not reached; colotomy in the left loin	Fatal, six days	No meconium escaped with the urine.
74	M	Guersant, ibid.	Imperforate anus; rectum terminating in the membranous part of the urethra	Incision and puncture, and gut reached	Fatal, six days	Wound in the peritoneum, and abscess in the pelvis, consequent on the puncture.
75	M.	Désormeaux, 'Gaz. Méd. de Paris,' 1856	Anus opening into a cul-de-sac; rectum terminating in a blind pouch in the pelvis	Puncture, and gut reached	Fatal, nine days	Difficulty in defecation, consequent on the upper portion of the rectum passing behind the anal cul-de-sac.
76	M.	Guersant and Guillot, recorded by Godard, 'Gaz. Méd. de Paris,' 1856	Imperforate anus; rectum wanting; colon terminating in a blind extremity at the brim of the pelvis, a small canal being prolonged to the base of the bladder	Incision, but gut not reached	Fatal, eleven days	Two attempts were made to reach the gut by operation.
77	F.	Guersant, ibid.	Anus opening into a cul-de-sac; rectum terminating in a blind pouch at the brim of the pelvis	Puncture, but gut not reached	Fatal, two days	—
78	M.	Lepestre, Acad. de Med., ibid.	Imperforate anus; rectum terminating in the pelvis in a blind pouch	Incision, but gut not reached; colotomy in the groin	Successful	Died from cholera at the age of fourteen months.
79	M.	Goyrand, "Etudes Pratiques," &c. ibid.	Imperforate anus; rectum opening at the scrotal raphé by a contracted outlet	Aperture enlarged by an incision at the age of six months, and border of intestine secured to the skin	Successful, sixteen years	Difficulty of defecation increased after birth until an operation became necessary. Patient alive at sixteen years of age.
80	F.	Goyrand, ibid.	Imperforate anus; rectum opening at the posterior commissure of the vulva by a contracted outlet	Aperture enlarged at the age of eleven months	Successful, six months	Died of a cerebral affection six months after the operation.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
81	M.	Goyrand, "Etudes Pratiques," &c., 'Gaz. Méd. de Paris,' 1856	Imperforate anus; rectum terminating in a blind pouch as low as the prostate gland	Incision, and gut reached	Fatal, thirteen days	Defecation free; died after thirteen days of thrush (muguet).
82	M.	Goyrand, ibid.	Imperforate anus; rectum terminating in the prostatic part of the urethra	Colotomy in the groin	Successful	Died of infantile cholera at the age of ten months and a half. Pelvis imperfectly developed.
83	M.	Thiry, 'Presse Médicale Belge,' Mai 8, 1859	Imperforate anus; rectum terminating in the urethra or bladder	Incision, and gut reached	Successful, four years	Tendency to close, and dilatation continued for eight months. Doing well at the end of four years. Fæcal matter occasionally passing by the urethra; no incontinency; anal aperture rounded, and plaited as in natural condition.
84	M.	Friedberg, 'Archives Générales de Med.,' 5ème série, tom. x	Imperforate anus	Incision, and gut reached, drawn down and secured in anal region	Successful, ten months	Lived ten months, and died from dentition.
85	M.	Friedberg, ibid.	Imperforate anus; the rectum opening in the perineum behind the scrotum by a narrow fistulous channel	Incision, and gut reached, drawn down, and secured in anal region	Successful, three months	Operation performed at the age of two months; died from pneumonia three months afterwards.
86	M.	Nagel, quoted by Friedberg, ibid., 5ème série, tom. ix	Imperforate anus; rectum communicating with the urethra, which terminated in front of the scrotum	Incision, and gut reached	Successful	Doing well at the end of several months; urine alone passing by the urethra, and fæcal matter by the anus.
87	F.	Nagel, ibid.	Imperforate anus; rectum opening into the vagina	Incision, gut reached and attached to skin at the anus	Successful	Operated on at the age of six months, in consequence of insufficiency of vaginal opening and obstinate constipation.

88	M.	Flajani, quoted by Friedberg, ibid.	Imperforate anus; rectum terminating in membranous part of the urethra	Incision at the age of seven months, but gut not reached	Fatal at the age of eight months	Fæces passed by the urethra, but child suffered great distress, and died from a cherry-stone blocking up the opening.
89	M.	Jenner, 'Path.Trans.,' vol. i	Anus opening into a cul-de-sac; rectum wanting	Puncture, but gut not reached	Fatal, ninth day	——
90	F.	Johnson, ibid., vol. xi, 1860	Anus opening into a cul-de-sac; rectum terminating in a blind pouch half an inch from the anus	Puncture, but gut not reached; colotomy in the groin	Fatal, two days after second operation	In the first operation the recto-uterine pouch was opened.
91		Johnson, 'British Med. Journal,' Jan. 7th, 1860	Imperforate anus; rectum opening into the vagina	Incision, and gut reached	Successful, several months	When the patient was last seen the passage was free. No attempt had been made to close the recto-vaginal opening, which was very small.
92	F.	Johnson, 'Lancet,' vol. i, 1860	Anus opening into a cul-de-sac three quarters of an inch in depth; rectum terminating in a blind pouch near the septum	Puncture, but gut not reached; colotomy in the groin	Fatal, two days	Peritoneum wounded by the puncture, and escape of serum from the cavity.
93	M.	South, 'St. Thomas's Hospital Reports,' vol. i	Imperforate anus; rectum opening in front of the scrotum by a narrow fistulous channel	Incision, and gut reached	Successful, eighteen years	Great difficulty in maintaining the opening, and operation repeated four times. A small quantity of fæces passed occasionally by the abnormal channel. Consent refused to an operation for its closure.
94	M.	Pretty, 'Med. Gazette,' vol. xlvii	Anus opening into a shallow cul-de-sac; colon terminating in a blind pouch at the promontory of the sacrum	Puncture, but gut not reached	Fatal, twenty-four hours	Death caused by over-distension, and rupture of ascending colon. Infant survived its birth eighty-two hours.

No.	Sex.	Operator and References.	Nature of Imperfection.	Treatment and Nature of Operation.	Result and Period.	Remarks.
95	M.	Leslie and Pennell, 'Med. Times,' vol. xxv	Imperforate anus; rectum wanting; colon terminating in a blind pouch	Deep incision, but gut not reached; colotomy in the left loin.	Fatal, thirteen days	Died of peritonitis, probably due to unsuccessful attempts to establish a passage at the anus several days after the operation in the loin.
96	M.	Mason, ibid., vol. xxvii	Anus opening into a cul-de-sac; rectum wanting; colon terminating in a pouch	Puncture, and gut reached	Fatal, twenty-four hours	——
97	F.	Ashton, 'Diseases of Rectum,' 3d edition	Imperforate anus; rectum terminating in a blind pouch one and a quarter inch from the surface.	Incision, and gut reached	Successful, three months	Two operations performed previously had failed, owing to great tendency to contraction. Operated on for third time at the age of four months. Died suddenly at seven months.
98	F.	Ashton, ibid.	Imperforate anus; rectum opening into the vagina	Incision, and gut reached	Successful, three years	Operation performed at the age of four weeks; great tendency to contraction, and use of bougie continued for two years. The recto-vaginal opening remains unclosed.
99	F.	Lane, 'Brit. Med. Jour.,' 1858	Imperforate anus; rectum opening into the vagina	Incision, and gut reached, and division of the septum between the two openings	Fatal, twenty-three days	Operated on at four and a half years of age. An operation performed at the age of twelve months had failed, and the wound had closed.
100	M.	Shaw, unpublished, communicated by operator	Anus opening into a cul-de-sac an inch in depth	Incision, and gut reached, drawn down and secured on one side of the natural anus	Successful, five years	Difficulty in maintaining the opening. Subject to distension of abdomen from accumulation of fæces.

Of these cases, 68 were males, and 32 females.

The congenital malformations of the rectum may be conveniently classed as follows:

1. *Imperforate anus, the rectum being partially or wholly deficient.*—Of this form, the table furnishes 26 instances—21 males and 5 females. Cases 1, 10, 13, 17, 18, 19, 22, 23, 27, 28, 32, 34, 36, 45, 51, 53, 56, 64, 67, 71, 76, 78, 81, 84, 95, 97.

2. *Anus opening into a cul-de-sac, the rectum being partially or wholly deficient.*—Of this, the table includes 31 cases—17 males and 14 females. Cases 2, 3, 6, 7, 14, 15, 16, 20, 24, 25, 26, 30, 31, 35, 37, 46, 47, 48, 49, 52, 54, 57, 60, 75, 77, 89, 90, 92, 94, 96, 100.

3. *Imperforate anus in the male, the rectum being partially or wholly deficient, and communicating with the urethra or neck of the bladder.*—Of this, the table contains 26 cases[1]—Cases 11, 12, 21, 33, 38, 39, 40, 41, 42, 43, 44, 50, 55, 58, 59, 61, 62, 65, 70, 72, 73, 74, 82, 83, 86, 88.

4. *Imperforate anus in the female, the rectum being partially deficient and communicating with the vagina.*—Of this, the table furnishes 11 cases—Cases 4, 8, 9, 29, 63, 68, 69, 87, 91, 98, 99.

5. *Imperforate anus, the rectum being partially deficient, and opening externally in an abnormal situation by a narrow outlet.*—Of this form the table contains 6 cases, 4 males and 2 females—Cases 5, 66, 79, 80, 85, 93.

A few other congenital deviations have been observed, but they are of very rare occurrence, and the five forms enumerated above are alone included in the table.

The following case occurred in my own practice: A male child, five weeks old, was brought to me on account of a congenital fæcal fistula communicating with the rectum. There was a depression at the back of the sacrum, about an inch and a half from the anus, with a small orifice through which a little feculent matter escaped, when discharges occurred from the natural orifice, which was of proper size. The fistula was too small to admit the passage of a common-sized probe.

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[1] It is very probable that 26 is less than the actual number, for the communication with the urethra is sometimes so minute as to prevent the escape of meconium during life, and readily to escape detection after death; a fact particularly noticed by M. Godard.

It will be observed that the classification of these imperfections is founded on states which can generally be recognised during life. Unfortunately the condition of the terminal portion of the intestinal canal, and its relations to the parts around, cannot be predicated with any certainty. In cases of imperforate anus, or of anus opening into a cul-de-sac, the intestinal canal may terminate in a blind pouch at the brim of the pelvis, the rectum being wholly wanting; or an imperfect rectum may form a shut sac, descending to the floor of the pelvis, or as low as the neck of the bladder in the male, or the commencement of the vagina in the female. It is known that the anal portion of the bowel is developed distinctly from the upper portion, and that the two afterwards approximate and unite, the diaphragm or septum disappearing by interstitial absorption. A failure in this process is the cause of the second form of congenital imperfection. The cases of imperforate anus in which the rectum communicates with the urethra or vagina depend on the original existence of a cloaca, the malformation being due to an incomplete separation during fœtal life. These conditions are the result of an arrest of development at different stages. The blind pouch, in which the intestinal canal terminates, is sometimes connected to the anal integument, or to the anal cul-de-sac, by a cord prolonged from the bowel above. These cases are not, like the preceding, the result of a non-formation of the rectum, but are produced by an obliteration of the bowel, which was originally well formed; the obliteration being a pathological change, due probably to inflammation which had existed during intra-uterine life. M. Goyrand, who has published some able papers on these malformations in the 'Gazette Médicale de Paris' (1856), quotes a case communicated by M. Forget to the Société Médicale d'Emulation, of a female infant with an imperforate anus, who died after an operation in the perineum, without the bowel being reached. The intestine terminated at the base of the sacrum in a pouch, from which proceeded a good-sized cord to be implanted in the skin at the site of the anus. This cord was formed of fibres continuous with the longitudinal fibres of the muscular tunic of the intestine. Friedberg also mentions a case in which the obliteration was less complete than in the preceding, of a female with an anus well formed, but with the rectum closed at a short distance above. At the autopsy the walls of the intestine were found adherent to each other in different places.[1]

The relations of the peritoneum to the bowel in the different forms of atresia have an important bearing on the operations performed in the perineal region; for there can be no doubt that in many instances, as in Cases 6, 30, 74, 90, 92, and others in the table, the fatal result was in some measure due to the opening made in the serous sac. When the rectum, being only partially defective, descends into the pelvis, as commonly happens in the third, fourth, and fifth forms, the peritoneum is reflected from the bowel at a distance from the part liable to be opened in the ano-perineal operation. But when the rectum is wholly wanting, the intestine not passing lower than the brim of the pelvis, the peritoneum completely invests the terminal pouch, and must necessarily be wounded before the bowel can be reached from the perineum. In cases of intra-uterine obliteration of the rectum, the peritoneum is also much exposed to injury. In Forget's case, quoted above, the serous membrane extended upon the fibrous cord for three lines, and was strongly adherent to it.

In cases of complete deficiency of the rectum, it has been remarked by Rokitansky, Goyrand, and others, that the pelvis is not well developed, the tuberosities of the ischium being near together, and the antero-posterior diameter abnormally small. A depression in the anal region, and the position of the genitals far back, would also lead us to infer an absence of the rectum. In some instances the pelvis is incomplete, as in Case 82, in which the sacrum was formed of only four false vertebræ, and wanted the coccyx. In several of the cases in the table the operators, finding the pelvis small, and other evidence of the rectum being wholly deficient, took the bold course of opening the colon in the abdomen, without adding to the dangers of the case by a deep incision in the ano-perineal region, and a search for the bowel without a reasonable hope of success.

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[1] 'Archives Générales de Médecine,' v^e série, t. ix, p. 569.

1. In imperforate anus the rectum may, as I have already stated, be entirely wanting, the colon terminating at the brim of the pelvis, or, it may be, only partially defective. It is rarely found at a less distance than an inch from the perineum. The 26 cases in the table furnish the following results:—In 14 cases the gut was opened in the anal region, and in 12 the operator failed to reach it. Of the former, 9 ended fatally[1] and 5 proved successful. Of the 12 cases in which the gut was not reached, 2 ended fatally, without anything further being done. In 7 colotomy was performed in the groin, one of which only proved fatal. In 3 the colon was opened in the lumbar region; one recovered and 2 died.

Of the 5 cases in which the gut was reached in the perineum, and an early death prevented, the following results were ascertained. In Case 32 the rectum appears to have been only partially defective, and the anus first made at the side of the coccyx being insufficient, and becoming obstructed by a plum stone at six years of age, another was formed in front with success. In Case 71 the rectum descended low in the pelvis, yet after the operation by incision there was great difficulty in maintaining a free passage, and the operation had to be repeated twice; but the child was alive and well at six years of age. In Case 97 the gut was reached by incision at the age of four months, after the failure from contraction of two previous similar operations; the child died suddenly three months afterwards. In Case 84, the rectum being partially defective, the rectum was not only incised, but drawn down and secured to the skin in the anal region. The infant died from the troubles of dentition at the age of ten months, but defecation was perfectly satisfactory up to that period. In Case 34 a similar operation was performed, except that the bowel was drawn down the day after it was punctured, and was not secured by ligatures to the skin. The published report of this case gives no information later than a month, but Dr. Smyth, of Chelsea, who assisted in the operation, has informed me that the result was perfectly successful to the age of eighteen months, when he lost sight of the case. This is the only instance of complete success, in which there is every reason to suppose that the rectum was wholly wanting. In the three cases, 32, 71, and 97, in which the bowel was simply incised, more or less difficulty was experienced afterwards in maintaining a free passage for the fæces; but in Cases 34 and 84 subsequent contraction was prevented by drawing the bowel down to the anal region.

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[1] Case 81 is included in the list of fatal cases, the infant having survived only thirteen days; but it is reported that defecation was free, and that death was caused by *thrush.*

2. When the anus opens into a cul-de-sac, the pouch varies in depth from half an inch to an inch and a half. Its fundus may be separated from the blind extremity of the rectum by a septum composed only of the two coats of the bowel and a little areolar tissue, or the upper part of the rectum may be wanting altogether or nearly so, a space more or less considerable intervening. The upper and lower portions of the rectum usually meet end to end, but this is not constant; for in Case 7, observed by myself, in Case 24, described by Amussat, and in Case 75, recorded by Godard, the lower portion ascended in front of the upper, so that for a short distance the two passages ran parallel. In Case 24 this arrangement was favorable to the result of the operation by incision into the bowel above, and division of the septum between it and the anal cul-de-sac. In Case 75 merely a puncture was resorted to; consequently the collection of fæcal matter in the upper or posterior pouch pushed the partition forwards, so that it formed a valve, which prevented the escape of the meconium without the introduction of a tube, a difficulty which led early to a fatal result. Case 20, recorded by Amussat, though included in

this category, does not strictly belong to it. It is remarkable from the circumstance of the natural anus opening into the vagina, both portions of the rectum being deficient. I know of no similar case.

In 16 of the 31 cases of the second form, the gut was reached and opened. In 11 the operator failed in finding it. Of the former, 6 were fatal and 10 recovered. In one of the fatal cases (52) the infant survived six months, but the surgeon was unsuccessful in maintaining the passage. Of the 11 cases in which the gut was not reached, 6 ended fatally without any further operation. In 2 colotomy was performed in the groin, with a fatal result. In 3 instances the colon was opened in the loin; 2 were fatal and one recovered. In 4 cases colotomy was performed without any previous subpubic operation; three times in the groin with successful results, and once in the loin with a fatal termination.

In analysing the 10 cases of success from a subpubic operation, I may dismiss Cases 35 and 49, as the dates of the results (two weeks, and four weeks) are too short to admit of satisfactory conclusions being drawn from them. In case 57 there appears to have been a septum, consisting of the coats of the two portions of the rectum in close apposition. This partition was incised, and a free passage readily established. The use of bougies was continued for seven weeks, and, as might be anticipated in so simple a case, the result was quite satisfactory up to the last report, two and a half years after the operation. In Case 54 the two portions of the bowel were evidently in close contact, and the septum thin. It was punctured by Sir Philip Crampton with a large trocar, and well dilated afterwards. All treatment was discontinued after six months, and at four years of age, when the child was well grown and in excellent health, a commission of eminent Dublin surgeons examined him, and reported that, at a distance of about an inch and a quarter from the anus, the fingers encountered a diaphragm, with an annular perforation, through which its extremity could be passed by a moderate amount of pressure. This remnant of the septum consisted of a narrow, circular slip of yielding membrane, the central opening occupying four-fifths of the calibre of the gut. The rectum was somewhat contracted at this point, and there seemed to be a slight deviation from its natural direction, the two portions meeting each other so as to form an angle slightly salient posteriorly. The passage of the fæces was unattended with pain or difficulty. In Case 47, also, the septum, so far as we may judge from the brief and imperfect report of it, appears to have been slight, as in the preceding case, and to have been readily perforated with a trocar. The opening was fully stretched with the finger and the passage frequently and thoroughly dilated for several weeks, when, as it showed no tendency to contract, this practice was discontinued. The infant thrived for a time, but died at two years of age, of croup. In Case 24, in which, as I have already stated, the rectal pouch descended behind the anal, Amussat incised the septum freely. I can find no report of the result later than eight weeks, but there is no reason to doubt its complete success. In case 20, in which the anus opened into the vagina, it was an important point, in order to prevent the entry of the fæces into it, to establish a new and distinct passage. Amussat made, therefore, an incision behind the anus, and the rectum being wanting, penetrated deeply (two inches), until he reached the bowel, which he detached, dragged to the perineum, and secured to the skin by sutures. The child subsequently came under the care of Sir Philip Crampton, who had to enlarge the opening at the end of two months. The ultimate result was perfectly satisfactory. The patient grew up and married at the age of twenty-one. Though the operation succeeded and the patient retained the fæces perfectly well, I agree in the opinion since expressed by Amussat, that in a similar case it would be much more satisfactory to unite the extremity of the intestine to the normal anus. Thus, in Case 100, for some brief notes of which I am indebted to Mr. Shaw, he attempted a similar operation to that practised by Amussat. The blunt end of the bowel, an inch deep, was drawn down, opened, and stitched to each side of the perineal incision. Whether owing to the artificial anus being established at one side of the natural anus, or to the small size of the opening made into the bowel, prolonged treatment was necessary to counteract the tendency to contraction which still existed when the boy was last seen, at the age of four or five years, at which time he was subject to great accumulation of fæces. In Case 3, I also adopted the plan of bringing down the bowel and securing it to the borders of an external incision, with a satisfactory result.—A male infant, five days old, was brought to the London Hospital, April 16th, 1860, in consequence of sickness and inability to pass the fæces. He was weak and emaciated, and had not slept for twenty-four hours. Several doses of castor oil had been given to him. The anus was well formed, but on passing a director and afterwards my finger, I found it to open into a cul-de-sac, three quarters of an inch in depth. A piece of sponge having been inserted and kept in for half an hour, in order to dilate the sac, I enlarged the anus by an incision towards the coccyx, dividing the posterior wall of the anal pouch to the depth of nearly an inch, when the upper portion of the rectum was opened, and meconium escaped. The bowel was seized with the forceps, drawn down, and secured to the wound in the skin on each side by two sutures. After the operation the infant took the breast well, slept, and passed its motions freely. The sutures were removed on the 21st. May 3d, though defecation was unimpeded, a bougie was passed for the first time, and, as a matter of precaution, directed to be introduced every other day, the size being soon increased to a No. 1 rectum-bougie. At the last date, October 4th, the infant, a remarkably fine child, passed its motions freely, a No. 4 rectum-bougie being introduced occasionally.

Case 37 is one of much interest, as illustrating in a striking manner the difficulties of treatment and the serious inconveniences so liable to arise after the formation, simply by incision or puncture, of a communication between the two portions of the rectum, when an interval of any extent exists between them. The cul-de-sac was only half an inch deep, and in order to reach the upper portion of the bowel, Mr. Le Gros Clark, the operator, had to carry his knife to the depth, including the sac, of two and a half inches before the gut was fairly opened. To preserve the passage, the thickened and contracted tissues required division at the end of three weeks. For several months the infant suffered from constipation and diarrhœa, with great distension of the abdomen, and about ten months after the second operation he again divided an obstructing band with a hernia-knife. Dilatation was afterwards persevered in. The boy is now nine years of age, in tolerable though not robust health, but subject at times to serious troubles in defecation.[1]

3. In the third form in which the anus is imperforate, the bowel opening into the urethra or bladder, the rectum is seldom entirely deficient, but the communication with the urinary passage at the neck of the bladder, or more commonly at the anterior part of the prostatic portion of the urethra, is always small and insufficient, and not usually direct, but takes place by a narrow canal. The meconium escapes consequently with more or less difficulty, and retention occurs at an early period. Liquid meconium may pass readily at first, but as the fæces acquire consistency, obstruction arises and life becomes endangered. There are a few cases on record in which, the communication being more free than usual, life has been preserved for many months, the fæces escaping entirely by the urethra, until, the passage becoming at length blocked up, death has ensued. This happened in two of the cases in the table, in which the operator failed in reaching the bowel. In one, Case 42, great distress resulted, but the child survived eight months and twenty-two days, death being partly caused by ulceration of the urethra and infiltration of urine. In the other, Case 88, the infant suffered considerably until the opening into the urethra became blocked up by a cherry stone, and death took place at the age of eight months.

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[1] I am indebted for the history of this case, since the report in 'The Lancet' (March, 1851), to the kindness of Mr. Armstrong, of Gravesend, and Mr. Le Gros Clark.

Dr. Steel has recorded a case in which no operation was performed, and the child lived eleven months, when the passage became obstructed by two large apple seeds, which caused death :[1] and Dr. Lyell, of Dundee, met with a case in which the child lived twelve months before fatal obstruction occurred.[2] The opening into the urethra appears to be usually of a valvular character, so that, although the fæces can pass into the urinary canal, the urine is unable to enter the rectum.

As the rectum is seldom entirely deficient, the bowel is generally reached with greater ease and certainty in this form than in the two preceding, and the results consequently are somewhat less unfavorable. Of the 26 cases in the table, the gut was reached in 15. In 9 the surgeon failed to find it. Of the former, 9 recovered and 6 proved fatal. In one of the latter the fatal result was delayed for ten months; in a second, thirteen months. In this case (55) the child died from a fall, but it is placed in the fatal list because the artificial opening had closed, and death must ultimately have ensued from the insufficient passage. Of the 9 cases in which the gut was not reached, 7 ended fatally, without any further operation. In one colotomy was performed in the groin, in the other in the loin; both ended fatally. In 2 cases no attempt was made to reach the bowel from the perineum, but the colon was opened in the loin. One did well, the other died.

In 8 of the 9 successful cases the bowel was opened simply by an incision, the operation having been completed in one of the instances with a trocar. In one, the details of which are only briefly given, the result is reported as satisfactory at the end of several months. In all the other 7 cases more or less difficulty was experienced afterwards in maintaining the passage, and in 3 the contraction was so great as to render a repetition of the operation necessary. In Case 40 the bowel was re-incised ten times before the age of eight months. In Case 39, the only one in which, after incision of the bowel, the walls were drawn down and secured to the skin, though dilatation was required for some time afterwards, no difficulty was experienced, and the patient was well and thriving at five years of age.

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[1] 'American Journal of Medical Sciences,' vol. xv.

[2] 'Edinb. Monthly Journal,' August, 1847.

After the establishment of a passage at the anus, the escape of fæces by the urethra did not always cease, and in some instances serious inconveniences resulted from non-closure of the abnormal communication. In Case 70 meconium escaped through the urethra only on one or two occasions, when the artificial channel became obstructed; and in Case 38 none passed after the first year. But in Case 83 fæcal matter was discharged occasionally by the urethra, at four years of age; and in Case 59 the patient was subject, at six years of age, to pain in the bladder and difficult micturition. In Case 43, in addition to the occasional escape of liquid fæces by the urethra, and of urine by the anus, on several occasions small, hard concretions were discharged from the bowel, and the operator, Mr. Fergusson, had to extract one about the size of a hazel nut. Small seeds, barley-pickles, and a small portion of bone, which had been swallowed, were also extracted from the urethra. I might have included in the table, had I noticed it in time, a case of the third form, operated on by incision, by M. Willaume, de Nancy. An anus was established with difficulty at the proper site, and dilatation was not abandoned until the age of twenty-eight. The young man, when seen by M. Willaume, at the age of thirty-one, was in good health, and passed his fæces without difficulty. He had suffered from abscesses in the scrotum, one of which had been caused by a plum stone swallowed four months before.[1] In Case 40 the bladder was wounded in the last operation, ever since which the urine was partly discharged by the anus. A very large alvo-urinary calculus gradually formed in the bowel, and was extracted with difficulty, by operation, at seven years of age.[2]

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[1] 'Gazette Médicale de Paris,' 1856, p. 526.

[2] Dr. Marcet ('Calculous Disorders,' 2d edit., p. 135) has given an account of a calculus the size of a walnut, composed chiefly of the phosphates found in the rectum of an infant born with an imperforate anus, the bowel communicating with the urinary organs.

4. In the fourth form, imperforate anus, with the rectum opening into the vagina, the bowel, after descending well into the pelvis, curves forwards and terminates in an aperture, which is generally pretty free, so that an operation is usually performed to remove a disgusting infirmity, and not for the preservation of life. In several instances, persons born with this imperfection have passed through life, submitting to the annoyances consequent upon it, and have married and borne children. Morgagni mentions the case of a woman who lived to the age of a hundred years, and passed all her fæces by the vagina. The communication, however, is not always sufficient, and the bend in the bowel seems unfavorable to the free escape of its contents. In Case 9, which fell under my own observation, there had been occasional constipation; and on examination of the child, who died at the age of four years, a month only after the operation for an artificial anus, a very dilated and hypertrophied rectum fully proved that an impediment had existed during life. In Cases 68 and 87 operations were resorted to at the age of six months, in consequence of the infants suffering from tenesmus and obstinate constipation; and in Case 99 at a still later period, after great mishief had taken place.

As the rectum descends low in the pelvis in this form, and as a curved probe can readily be passed into it by the vaginal opening, to serve as a guide for the incision, the operation for an artificial anus is attended with very little risk, and the surgeon cannot well fail to reach the bowel. In all the eleven instances the gut was opened, and only one ended fatally. In this case (99) the operation was a second one, and performed at four and a half years of age. The ill result was caused by excessive distension of the rectum previous to the operation, which had been delayed too long by the parents to admit of a favorable termination. Eight of the remaining ten cases are reported as successful, and two as unsuccessful, owing to the tendency to contraction, and the neglect by the parents of the means recommended to maintain the passage. In Case 63 the patient was twice operated on by incision, but the artificial anus closed after both operations. Case 4 came under my own care, in 1848, and was operated on also by incision, before I was aware of the importance of attaching the bowel to the skin.—A female infant, three weeks old, was brought to me on account of an imperforate anus, and the passage of the fæces by the vagina. There was no trace of anus, only a faint depression in the mesial line. In the posterior wall of the vagina, about three quarters of an inch from the orifice, there was an opening, communicating with the rectum, capable of admitting a No. 8 bougie, through which the fæces were voided without difficulty. At the request of the parents, I made the attempt to establish a passage at the natural site, when the infant was a month old. With a narrow-bladed bistoury I cut down carefully until I reached the rectum, at about three quarters of an inch from the surface. I made a free opening, into which I directed the nurse to pass a good-sized bougie twice a day, retaining it half an hour each time. As the parents lived in the country, the infant was not brought to me so frequently as I wished. At the end of ten days I found that the bougie had not been passed deep enough to enter the bowel, and that contraction had taken place. With some difficulty I re-established the communication, and secured a tube there, but it slipped out during the following night. As I could place no dependence on the nurse passing an instrument properly, I persuaded the parents to send the infant to me daily, in order that I might perform dilatation myself. After a fortnight they allowed three days to pass without bringing the child, thinking that the nurse could manage matters. I now found that great contraction had taken place, and had considerable difficulty in again re-establishing the passage. This was effected by introducing a curved sound through the recto-vaginal opening, to serve as a guide for a bougie. I enjoined perseverance in dilatation, but the mother, getting tired of the constant attention requisite in the treatment, and harassed by the infant's cries, which were incessant during the presence of an instrument, discontinued the dilatation, and allowed the artificial anus to close. The child died a year afterwards from some disease of infancy.

Of the 8 successful cases, there is no account of Case 68 later than the 16th day, and of Case 69 later than five weeks; and in Case 9 the infant died of diphtheria in a month. Case 8 came under my notice in July, 1853, five months after the operation.—A healthy female infant, aged five months, was brought to me by the mother, who stated that after birth all the motions passed through the vagina. She took the child to St. Bartholomew's Hospital, where an operation was performed, since which the fæces had passed partly behind and partly in front. I found a slender band of integument across the perineum, behind which was the termination of the rectum. The opening was more forward than the usual situation of the anus, and unclosed by any sphincter, the mucous membrane being protruded. When defecation took place, the evacuation came in contact with the band, escaping partly behind it and partly in front, just at the entrance of the vagina. As this slender band interfered with the passage of the fæces, directing them to the front of the vagina, and as the opening behind was too extensive to admit of any hope of my being able, by an operation, to connect this part with the wall between the vagina and rectum, I divided it with a bistoury, thereby getting rid of a barrier to the free escape of the fæces externally, and leaving the termination of the rectum and of the vagina separated only by a thin partition of double mucous membrane. The subjects of Cases 29, 91, and 98, were operated on simply by incision. In Case 29 dilatation was continued at the last report, four months afterwards. In Case 91 a large bougie was passed without difficulty, when the infant was last seen, at the end of several months. In Case 98 there was great tendency to contraction, and the use of a bougie was continued for two years. In Case 87 Nagel cut upon the bowel, drew it down, and secured it to the skin, with a good result; but the report of the case, as given by Friedberg, is very brief.[1]

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[1] I have searched the Berlin journals for fuller particulars of this case, but have not succeeded in finding any.

In this malformation the establishment of a new passage at the natural site is not all that is required. We have also to obtain the closure of the abnormal communication with the vagina. In Case 87 the operator, eight days after forming an artificial anus, applied a hot iron to the margin of the recto-vaginal opening, in order to obliterate it, but without success. In Case 29 the opening is reported to have closed spontaneously, two months after the operation in the perineum. So fortunate a result is unlikely, and could occur only when the abnormal aperture is unusually small. In Case 91 the fæces, when liquid, passed occasionally by the vagina. I recently had the opportunity of examining the patient in Case 98, three years after the ano-perineal operation, and found that the fæces continue to escape freely by the vagina. I am not acquainted with any case in which, after the formation of an artificial anus at the proper site, a successful operation has been performed for the closure of the recto-vaginal opening; and Case 29 is the only one in the table of complete success; a new passage being established, and the abnormal opening closed.

5. In the fifth form the anus is imperforate, but the rectum opens externally, in an abnormal situation, by a narrow outlet. Four of the six cases in the table were males, and two females. In the males the opening was in the perineum, just behind the scrotum, in two; in one, in the scrotal raphé; and in one, anterior to the scrotum. In the females the opening occurred in the perineum, close to the vagina, or at the posterior commissure of the vulva. In all of the cases the vent was insufficient, and defecation more or less difficult. In Cases 79 and 85 the symptoms of retention, at the end of two months, became most urgent. The existence of an outlet for the fæces, though small and insufficient, prevents immediate danger to life; but sooner or later serious inconvenience arises. As the fæces acquire consistency, they escape with increasing difficulty. The bowel undergoes excessive dilatation, and constipation and retention at length ensue, and life becomes endangered. In this form, as in the last, the rectum can

generally be opened without much difficulty, for by introducing a director by the abnormal opening, the operator can scarcely fail in reaching the bowel. It was opened, therefore, in all six instances; though in one, Case 85, the communication between the outlet and rectum was a long, fistulous channel, and the bowel was deeply seated, so that the operation was difficult and protracted.

Two different operations have been practised to remedy this imperfection — 1, the enlargement of the original outlet, which was done in two instances; and 2, the establishment of a new anus at the natural site, which was performed in the four other cases. In Cases 85 and 93, the latter was the only proceeding practicable. In Case 85 Friedberg cut down upon the bowel, detached and drew it down to the external wound, and confined it to the skin by sutures. The result, so far as could be ascertained at the end of three months, was quite satisfactory. In Case 66 the child was operated on by incision, at the age of two years, but there is no account of the result later than six days. In Case 93, also, merely an incision was made into the bowel, and so great was the tendency to close afterwards that the aperture had to be enlarged by an incision four times, but the ultimate result was successful, though the original passage remained unclosed, and allowed occasionally a feculent discharge, consent having been refused to an operation proposed for its obliteration.

The following are the particulars of Case 5:[1]—In November, 1857, I saw, with Mr. Gardner, of Gloucester Terrace, a female infant, a few weeks old. He had noticed at its birth that the anus was remarkably small, and in advance of its usual situation, and he had found it necessary slightly to dilate the orifice. The infant was thin, feeble, and badly nourished, being brought up by hand. The abnormal outlet was small in size and close to the vagina, a slight septum only intervening. The anus was imperforate, but at its site there was a firm, oval elevation, with an indistinct depression in the centre. On examination with a bent probe introduced at the outlet, it seemed that the lower part of the rectum made a bend forwards from the sacrum, the convex part passing close to the integuments where the anus should open. Though the abnormal aperture was obviously insufficient, I did not like to interfere whilst the infant was in such feeble health, but I requested to be sent for in case of any retention taking place. In a few weeks the infant improved in health, but temporary obstructions occurred, and in the beginning of January, 1858, it became evident that the lower bowel was much loaded. There was much straining to pass the motions, and considerable prominence in the perineum; and although there was no actual retention, it was considered undesirable to delay an operation. A narrow, oval portion of skin only was dissected from the site of the anus, and the bowel was reached at the depth of only an eighth of an inch. Directly it was opened, solid evacuations were forced out, and a large quantity escaped. The opening was enlarged so as to admit an elastic-gum tube, cut from a No. 9 catheter, which was secured in the passage by elastic tape. The child went on well after the operation. The tube or a plug was passed daily, and kept in for a short time to prevent contraction. In a month the new anus was quite cicatrized. The motions passed by both apertures, but came away of larger size and more copiously from the artificial one. The child is now, two years and five months since the operation, thriving and well, with power of retention. I have delayed adopting any proceeding for closing the abnormal passage until the new one was thoroughly established, and no longer required dilatation. The parents reside in the country, and propose shortly to bring the child to me for the completion of the treatment. In this case the rectum descended so low in the perineum, that it was unnecessary to draw it down or to attach it by sutures to the skin.

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[1] A similar case, in which an artificial anus was established in the anal region, is related by M. Berard, 'Gazette des Hôpitaux,' 1844, p. 286.

The enlargement of the original outlet was performed in Cases 79 and 80 by M. Goyrand, who strongly advocates this operation in preference to the formation of an artificial anus at the natural site. In Case 79 the rectum opened in the perineum, behind the scrotum, by a contracted orifice. At six months the operator enlarged the aperture by an incision carried into the perineum, and afterwards secured the borders of the divided intestine to the cut surface of the skin by sutures. At sixteen years of age defecation was free, and power of retention complete, even when the motions were lax. In Case 80, a female, the rectum opened by a narrow orifice at the posterior commissure of the vulva. At the age of eleven months M. Goyrand enlarged the aperture by an incision towards the coccyx, and applied sutures, as in the former operation, with a satisfactory result, but the child died, six months afterwards, of a cerebral affection. The chief advantage of this simple operation is the security against subsequent contraction; for as the upper angle of the opening is untouched, the lower one only has to undergo cicatrization, and, though the aperture does not come within the influence of the sphincter muscle, continency, it is said, is preserved. There is also no necessity for the performance of a second operation to close the abnormal opening, which becomes necessary when a new and distinct outlet is established. Larger experience is necessary to determine which of the two is the better operation. If the rectum were deeply seated in the anal region, there would be much force in the reasoning which induced Goyrand to enlarge the abnormal outlet, but we know, on the contrary, that the bowel is generally near the surface. In Cases 5 and 85 an artificial anus was easily established at the proper site, and, though difficulties occurred in Case 93, it is most probable that they would have been avoided had the bowel been secured to the skin at the time of the operation. Besides, in Goyrand's second case, a female, the anus was left in inconvenient proximity to the vagina, and we may apprehend that in the event of pregnancy taking place, there would be great risk, during labour, of rupture of the frail barrier between the two passages.

In cases of imperforate anus in which a passage is successfully established, the retentive functions of the bowel generally exist in sufficient force. We have satisfactory evidence on this important point in several of the cases in the table (Cases 5, 32, 37, 38, 40, 43, 83), and the existence of an external sphincter, at the natural site of the anus, has been frequently recognised in dissection (Cases 30, 70, 74, 81) in the form of a band of parallel fibres, situated in the median line, without any central separation. But in Case 82, in which the last bone of the sacrum and the coccyx were wanting, and in another case of absence of the coccyx, referred to by Goyrand, the sphincter was very imperfectly developed, its fibres being few and slender.[1] As in this imperfection the external sphincter consists of a single straight muscle, the surgeon, in operating, should be careful to make his incision exactly in the median line, so that the fibres may be separated equally, and the new anus be brought fully under the influence of the muscle.

In cases of imperforation, unremedied by operation, death is sometimes caused by extreme distension and rupture of the colon, or terminal pouch. In Case 94, in which the operator failed to reach the gut, the infant died in eighty-two hours; and at this early period the colon was found ruptured. My old pupil, Mr. Lys, of Bere Regis, sent me the parts, in a case of imperforate anus, unoperated upon, in which rupture of the pouch, and extravasation into the peritoneal cavity, occurred on the fourth day after birth. The accumulation of meconium, and distension of the colon, vary a good deal in infants, as I have often observed in post-mortem examinations; and operations have been performed, with success, much later than the period of death in the instances just quoted. In Case 3, the infant was five days old, when I opened the rectum, and saved life. There are, indeed, some remarkable cases of vitality under complete obstruction on record. Dr. Lyell, of Dundee, met with a case in which an infant lived upwards of twelve weeks, without any fæcal outlet but the mouth.[2] Although, therefore, I have omitted to note in the table the period after birth, at which operations have been performed, there can be no doubt that in many instances they were undertaken at too late a period to obtain success. The second form in which, with a well-formed anus, the rectum is imperforate, is very apt to be overlooked by nurses, and even by medical men, and not to be discovered in time to admit of life being saved by surgical treatment.

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[1] In Case 53 in the table, Mr. R. Davies was unable to discover any muscular fibres at the anus after death.

[2] 'Edinb. Monthly Journal,' 1857.

The most common causes of death after operation are peritonitis and diffuse inflammation of the areolar tissue. The former is generally produced by a wound of the serous membrane; the latter, in infants enfeebled by want of rest and nourishment, by the passage of fæcal matter through the tissues of the pelvis. These ill results are chiefly due to faulty methods of operating. In the cases in the table, mentioned as treated by puncture, a trocar was used, but this is a most unsafe instrument. A thin, bulging septum, at the end of an anal cul-de-sac, with a fluctuating feel, may be perforated in this way without much risk, but even in this case an incision is preferable. An operation for imperforate rectum should be conducted with the same caution and the same care as an operation for strangulated hernia; and the plunging of a trocar or a bistoury into the depths of the pelvis, in the faint hope of its penetrating the rectum, but at the risk of wounding the peritoneum, the bladder, or other important parts, is a rash proceeding, condemned both by reason and experience.

The plan of drawing down the bowel, and securing it by sutures to the margin of the wound made in the skin, was first performed, I believe, by Amussat, in Case 20, in 1835. This mode of proceeding has also been particularly described and recommended by Dieffenbach.[1] In comparing the cases in the table, operated on in this way, with those treated simply by incision, the great superiority of the former is strikingly apparent, especially when the bowel is at any distance from the surface. The important advantage obtained by it is the securing a lining of mucous membrane for the passage traversed by the fæces. By this means we not only guard against the tendency to contraction, with its consequent miseries and dangers, but also avoid the early risks of inflammation and fæcal absorption.

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[1] 'Die Operative Chirurgie,' 1845-8.

In some instances troubles in defecation have continued after a sufficient passage for the fæces has been fully established. This is owing to an organic change in the bowel, consequent upon an obstruction of long continuance, subsisting after the removal of the cause. In imperforation the rectum becomes more or less distended, sometimes in a remarkable degree: yet if an outlet of sufficient size be obtained early, the dilatation subsides, and the bowel recovers its natural size and tone. But when the passage or aperture is too small, or subject to recurring contraction, the bowel undergoes changes analogous to those observed in ordinary stricture of this part. In Case 9 the opening into the vagina was insufficient, and the child died at the age of four years, a month only after the formation of an artificial anus. The rectum was examined for me by Dr. A. Clark. It measured 5¾ inches in circumference in its most dilated part. Its longitudinal muscular coat was red, greatly developed, and columnar, like the fleshy columns of a hypertrophied bladder. The circular muscular coat was also red and highly developed. The mucous coat was covered with a thick, tenacious mucus, and studded closely with the openings of enlarged follicular glands. In the museum of St. Bartholomew's Hospital there is a similar specimen taken from a child who lived two years after the puncture of the rectum. The insufficiency of the aperture is proved by the great distension, the thick muscular walls, and the large follicles of the rectum above it. Case 99 furnishes a remarkable example of excessive distension and hypertrophy of the bowel consequent upon long-continued obstruction. The infant was born with an imperforate anus and a small recto-vaginal fistula, through which the fæces passed until the age of four and a half years, when the bowel was opened at the anus, and the septum between the two apertures divided. Death ensued in twenty-three days. It is stated that "the continual strain upon the rectum had distended it to a size of which it is hardly possible to give an adequate description." The rectum and sigmoid flexure of the colon formed an immense reservoir capable of containing five pints of fluid, which occupied the pelvis, the hypogastric, both iliac, and part of the umbilical regions, and pushed the viscera upwards, diminishing the cavity of the thorax.

When the vent for the fæces has long remained insufficient, and the bowel has undergone the changes above described, its expulsive functions become seriously impaired and weakened, and the infant consequently suffers in the same way as adults labouring under stricture of the rectum. The patient is subject, as in Case 100, to great accumulation of fæces, and the fæcal collection often sets up mucous irritation, which ends in diarrhœa. Mr. Le Gros Clark has given me an account of the present state of the patient in Case 37, which well illustrates the symptoms in this condition of the bowel. Great difficulty was experienced for some time in keeping the artificial passage free, and three operations were performed to relieve obstruction; but now, at nine years of age, though this difficulty has ceased, there is a very serious one of a different character. He says, "As far as I can judge by examination with the finger, the cul-de-sac, or sacculated extremity of the bowel above the artificial passage, has become gradually more distended under the pressure to which it has been subjected by the accumulation of feculent matter therein. Long intervals elapse without any attempt at relief; and, finally, a crisis arrives, after a fortnight, or even longer, when purging carries off the contents of the bowel, the quantity, of course, being very considerable."

Having, in the preceding observations, investigated the results of the operations performed in the anal or perineal regions, I have still to inquire more particularly into the degree of success which has followed the operations for opening the colon in the groin and in the loin, to ascertain the inconveniences consequent upon an anus made in these regions, and to estimate the comparative value of the two operations.

Colotomy was performed in 21 of the cases in the table, in 14 by Littre's, or the inguinal operation, and 7 by Callisen's, or the lumbar operation. Of the 14 cases in which colotomy was performed in the left groin, in 9 an unsuccessful attempt had previously been made to reach the gut from the perineum. Of these 4 proved fatal and 5 recovered. In 2 of the fatal cases (90 and 92) it is stated that the peritoneum was wounded in the first operation; and in a third (72), that the patient died of peritonitis and abscess consequent upon the puncture in the perineum. Of the 5 cases in which no operation had been performed in the ano-perineal region, one only proved fatal, and 4 recovered.

Of the 9 recoveries after the inguinal operation, one (Case 18) survived only a month; 2 died of cholera, one (Case 82) at the age of ten months, and the other (Case 78) at fourteen; a fourth (Case 64) was doing well at seventeen months; a fifth (Case 19) survived three years, and a sixth (Case 16) was doing well at thirteen years of age. Of the remaining three we have an authentic report in an interesting memoir recently communicated to the Imperial Academy of Medicine at Paris, by M. Rochard, a naval surgeon and professor at Brest, where the cases occurred. In Case 10 the patient died at the age of forty-three of a disease unconnected with the infirmity. In Cases 14 and 15, the subjects of the operation were still alive and well, the first at forty-six years of age, and the second at forty-three.

Of the 7 cases in which colotomy was performed in the left loin, an unsuccessful attempt had previously been made to open the bowel from the perineum in 5, of which 3 were fatal; and in one instance (95) a fruitless attempt was made several days after the lumbar operation, and the infant died of peritonitis. In Case 28 colotomy was delayed till the thirteenth day. In another fatal case (73) two unsuccessful operations had been performed in the perineum. In Case

6, which came under my care some years ago, there was an anus opening into a cul-de-sac. As a deep puncture had already been made through the parts at the end of the sac without reaching the bowel, I performed colotomy in the left loin. The kidney interfered; but the colon was reached, and opened without wounding the peritoneum, and a free discharge of meconium followed. The infant died eighteen hours after the operation; and, on examination of the body, I found that the rectum was entirely wanting, and that the colon terminated in a blind pouch in the left iliac fossa. The instrument introduced at the anus had penetrated the vagina, passed behind the uterus, and wounded the peritoneum. In the only case (26) in which colotomy was performed in the loin without a previous operation in the ano-perineal region, the infant died in eight days.

The inquiries instituted into the unsuccessful cases of colotomy, both in the loin and in the groin, leave no doubt that the fatal results were due in a great measure to the injuries inflicted in the attempts which had been made to establish an anus in the perineum.

Of the two recoveries after lumbar colotomy, in Case 25 Dr. Alphonse Amussat, the son of the operator, reports that the infant lived to the age of seven;[1] of Case 27, I can obtain no report more recent than seven weeks, and I have been informed that the child did not long survive the operation.

In endeavouring to arrive at a just conclusion as to the value of the two methods of performing colotomy, these operations must be studied and compared in reference chiefly to three questions: 1. The difficulty of the operation. 2. Its dangers. 3. The condition of the artificial anus and the inconveniences attending it.

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[1] 'Journal of Practical Medicine and Surgery,' Paris, March, 1859, p. 105 (English edition). Dr. A. Amussat states that his father performed this operation upon a child in May, 1852. The patient is now living at the age of eight years, in good health, and properly performing its digestive functions. This case has not yet been published. It seems that Amussat had only two really successful cases in infants.

1. The operation of making an artificial anus is admitted to be, under ordinary circumstances, one of greater difficulty in the loin than in the groin. This arises from the greater depth at which the colon is situated in the lumbar region, and the hardness of distinguishing the bowel when it is exposed. But there are several circumstances which are liable to add greatly to the operator's difficulties. Some of them are well portrayed in Amussat's description of his first two operations in infants. In a great fat child the depth is so considerable, that a free incision is required to reach the colon. The kidney varies a good deal in size at this period of life, and when large it overlaps and conceals the bowel. Again, the colon, instead of being distended with meconium, as might be expected, is sometimes contracted, and very hard to find. There are, besides, irregularities in the position of the colon which render it impossible to open the bowel in the left loin without wounding the peritoneum; and there are some which also prevent the operator from finding the colon in the left groin. With the view of gaining information on these important points, I practised both these operations on the bodies of 20 infants, 18 stillborn, and 2 deceased a few days after birth; and I afterwards examined the position and course of the colon in each subject. In 18 of the 20 subjects, I found colotomy in the left groin, whether the bowel was distended or not, an easy operation. In one of them the ovary and Fallopian tube protruded, but they were readily put aside and the colon reached. In two instances I was unable to find the colon. Both the subjects were well-formed infants, stillborn at the full term, one male, the other female. On opening the bodies I observed that the descending colon before reaching the pelvis made rather a sharp curve, and, passing across the abdomen in front of the fourth lumbar vertebra, formed an ample convolution on the right side before terminating in the rectum. Colotomy in the left groin was impossible, therefore, in these two cases, owing to the unusual course taken by the descending colon; but the bowel was easily opened in the right groin. This disposition of the colon has been observed

in a case of imperforate anus, one already referred to,[1] where death resulted from rupture of the terminal sac. Mr. Lys, who communicated the particulars of the case to me, states that the descending colon, instead of being directed to the left iliac fossa to form the sigmoid flexure, passed transversely across the spine over the fourth lumbar vertebra to the right sacro-iliac symphysis, and, descending into the pelvis, terminated in a cul-de-sac at the base of the bladder.

In 8 of the 20 subjects the colon was readily found, and opened in the loin without wounding the peritoneum. In 6 the operation was more or less difficult, owing in 2 to the great depth of the gut, in 2 to its being empty and contracted as well as deeply seated, and in 2 to a large kidney being in the way and concealing the bowel. Had the subjects been living, I apprehend that the difficulties of the operation would have been increased. In 6 subjects lumbar colotomy was impossible without opening the peritoneum, in consequence of the colon being attached by a distinct mesentery, and being loose in the abdomen. In three of the instances, I measured the meso-colon, and found it an inch in width. This serious impediment once occurred to me in performing the operation of lumbar colotomy in the living subject, in a case of imperforate rectum (Case 7), with the additional difficulty that the colon was empty and contracted, as well as loose in the abdominal cavity. Although deviations from the usual disposition of the colon may prevent or mar the success of colotomy in both regions on the left side, it would appear that this impediment is much more likely to be met with in an operation in the loin than in one in the groin; and if we add to this the other hindrances above mentioned, the difficulties liable to be encountered in the lumbar operation are certainly much greater and more serious than in the inguinal. A surgeon of common skill would find the latter an easy operation.

2. In respect to the dangers of the operation, the cases in the table are perhaps too few in number to enable us to

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[1] *Vide* page 305. This case has since been published by Mr. Hodges, of Rochford ('Lancet,' vol. i, 1859, p. 402).

make a satisfactory comparison between the two methods; but, so far as they warrant any conclusion, the results are much in favour of colotomy in the groin. Thus, if we except altogether those cases in which the peritoneum was wounded in an unsuccessful operation in the ano-perineal region, out of 11 cases of the inguinal operation, 2 only ended fatally; or, if we take the 5 cases in which no perineal operation was attempted, one only had a fatal termination, which gives a degree of success most encouraging to the surgeon. On the other hand, if we except 3 fatal cases of lumbar colotomy in which a previous perineal operation and other circumstances rendered recovery unlikely, we have 4 cases, of which 2 recovered and 2 died. In colotomy in the loin, though the abdominal cavity be unopened, the wound required is of greater extent and depth than in inguinal colotomy; and since, as I have already shown, the peritoneum is very liable to be wounded in consequence of the bowel being only loosely attached by meso-colon, or being empty and contracted, we should anticipate quite as much, if not greater, risk to life from this operation than from the inguinal; a conclusion confirmed by the results of the operations in the cases in the table. I believe, however, that the dangers arising from opening the peritoneum in these operations in the new-born infant are less than is commonly supposed.

3. The sufficiency of an anus, made either in the loin or in the groin, for its necessary functions, as well as its condition in relation to the comfort of the patient, are very important considerations, not only as bearing on the comparative value of the lumbar and inguinal operations, but also in reference to the question which parents have to consider, whether life is worth preserving with such an infirmity attaching to it. After the operation for artificial anus in the groin, the outlet seldom evinces any disposition to contract, and proves adequate for the passage of the fæces. M. Rochard has given an interesting account of its condition in several patients who had undergone the operation many years previously.

A robust woman employed in hard labour, operated on in 1813 (Case 14 in the table), had excellent digestion, and passed solid stools periodically. When defecation is about to take place, she is warned by a sense of inconvenience and fulness in the left flank. She then withdraws the bandage and compress from her body, and replaces it when the desire is satisfied. No fæcal matter passes in the interval, but a little moisture escapes at times from the upper part of the aperture. It is only when diarrhœa occurs that she is annoyed by the discharge of feculent matter.

A lady, operated on in 1816 (Case 15), constantly enjoys the best health, goes into society, and attends balls, and no one would suspect her to be the subject of any infirmity. She is married, has borne four children, and her pregnancies and labours have been quite normal. She never experiences the least pain in the part. In all the patients observed by M. Rochard, an inversion or prolapsus of the intestine, varying from about one to four inches, had taken place; and what is remarkable, the prolapsus occurred exclusively from the portion of the bowel beyond the artificial opening, owing, probably, to the circumstance that the colon above was fixed in the loin, whilst the part below was free and moveable. The prolapsus did not cause serious inconvenience, and by a well-fitting apparatus, making a certain amount of pressure, might be almost entirely prevented.

Of the condition of the anus in the lumbar region no satisfactory account can be obtained from the reports of the two successful cases in the table, one having died quite early, and the other at the age of seven years. In the latter case (25) it appears that there was difficulty in keeping the anus patent, and that life had twice been in danger from retention. Colotomy in the loin has been successfully performed so often in the adult to relieve obstructions in rectum, that we can form a tolerably correct opinion of the convenience of an anus in this region;[1] and, through the kindness of Mr. Walter Bryant, I have recently had several opportunities of seeing a boy, eight years of age, born with an imperforate anus, the rectum communicating with the urethra, whose life was saved by this operation. The case is one of much interest.[1]

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[1] I have myself performed lumbar colotomy in the adult four times, and in two of the patients who recovered no contraction of the artificial

The patient was born in South America in January, 1852. A German surgeon made an incision at the anus to open the bowel, but without success. He consequently performed colotomy in the left loin. The boy thrived, and in the early part of this year was brought to England. Mr. Erichsen was consulted as to the propriety of an attempt being again made to establish an anus at the natural site, the operation having been recommended; but he declined to sanction it. I saw the boy with Mr. Bryant in the month of June. He was healthy, well developed, active, and remarkably intelligent. The site of the anus was almost natural in appearance, presenting the puckering produced by an external sphincter, but it was closed by skin. There was a cicatrix at the side, about two lines from the median line. The father informed us that the fæces escaped freely from the artificial anus, and had always done so since the operation. There had been no tendency to contraction. The boy had no sensation or warning before an action took place, which occurred usually at night. He was seldom inconvenienced by feculent discharges at other times, unless the bowels were relaxed. His chief trouble was painful and difficult micturition. This occurred at uncertain times, but had increased in frequency during the last two years, and he usually suffered from it two or three times during the week. It obliged him to strain violently in passing water, and caused altogether great distress. There was an inguinal rupture on the right side, which seemed to have resulted from the violent straining. The

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anus ensued, and defecation took place readily. By drawing the bowel to the surface, making a free opening into it, and attaching it to the outer wound, tendency to contraction is avoided, both in lumbar and inguinal colotomy.

[1] This case came under my notice too late to be included in the table.

admixture of feculent matter with the urine passed on these occasions showed that some portion of the fæces escaped occasionally into the colon beyond the artificial anus, and from thence into the urethra, obstructing the passage. He was obliged to be very careful in his diet, and to avoid swallowing currants and other indigestible matters. The anus was of ample size, and oval in shape, with the red mucous membrane constantly protruding in close-set folds. The surface of the membrane was not at all sensitive. The finger passed easily into the bowel, both above and below, and was slightly girt by the actions of the abdominal muscles around the aperture. The surrounding skin was free from irritation. A prolapsus of the bowel readily took place on the slightest straining, and usually occurred when the part was uncovered for the purpose of cleanliness. The protrusion was easily reduced by slight pressure; but on one occasion some months previously, a very considerable inversion occurred, and it was so difficult to replace, that chloroform was administered to effect the reduction.[1] The position of the anus at the outer part of the loin, three inches from the spine, did not render personal attention to it difficult or inconvenient, and the little fellow could easily replace the ordinary protrusion by slight pressure with a napkin or sponge, whilst a bandage giving gentle support was sufficient to restrain it at other times. He ran about and took exercise like other boys, and no one would suppose him to be labouring under any infirmity, until obstruction occurred in micturition. To prevent the occurrence of this very serious evil, two modes of proceeding seemed to offer. 1. The constant wearing of a plug in the lower portion of the colon to prevent the entry of fæces into it. 2. An operation for closing the lower opening into the colon. As the second plan would not be free from some risk, we agreed to make trial of the first; and a piece of sponge, of proper shape, secured with a strong ligature, was lodged in the colon below the outer opening. This seemed partly to answer the purpose, and to save the boy distress in micturition; but it did not entirely prevent the entrance of liquid fæcal matter, and the plan was not persevered in by the parents. The lower portion of the colon was, however, washed out daily by injections.

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[1] M. Larrey showed, at a meeting of the Société de Chirurgie de Paris, an infant seven months and a half old, born with an anal imperforation. M. Maisonneuve, having failed to reach the bowel in an operation in the perineum, performed colotomy in the loin with success. The anus was the seat of an extensive double prolapsus, and had a remarkable appearance, the ends of the inverted bowel diverging in opposite directions. No attempt had been made to reduce it. ('Bulletin de la Société,' t. vi, p. 410.)

In health an artificial anus is productive of much less personal annoyance than is commonly supposed. Some degree of protrusion of the mucous membrane of the colon will always be liable to occur in consequence of the deficiency in the abdominal wall, and the want of support at the part. In a sound condition of the digestive functions, the fæcal evacuation is regular and periodic, and the part is almost free from discharge at other times, whilst a well-fitting apparatus is sufficient to restrain protrusion, as well as to prevent unpleasant oozing from the part. When diarrhœa occurs, or unhealthy gases are generated, then the annoyances are considerable, and may render the patient unfitted for society. It has been argued that an anus in the loin is much more inconvenient than one situated in the groin, as regards personal attention, whilst one in the groin is likely to be more repulsive in the relations of adult life. I confess that I see very little to justify a preference for either operation, on the ground of the position of the anus; but the greater difficulties and dangers of lumbar colotomy, which I have already pointed out, would certainly induce me, in future, to select the inguinal operation.[1]

In a recent discussion at the Imperial Academy of Medicine in Paris,[2] the question has been raised, whether the left side should be selected for the inguinal operation. M. Huguier strongly insisted on the importance of performing colotomy in the right groin. He stated that during intrauterine life the sigmoid flexure is enormously developed, and finding itself restricted in the left iliac fossa, passes over to the right iliac fossa, and thence dips into the pelvis to join the rectum. This disposition is observed in children up to the age of eighteen months or two years. The opening, therefore, if made in the bowel on the right side, would be nearer its termination, and the infant would be less liable to prolapsus, in consequence of the retraction of the sigmoid flexure, as the infant grows. I practised colotomy on the right side in five infant subjects. In three the colon was easily reached, and, in two of these, the convolution of the colon, from the left groin to the right, measured nine inches in length. In a fourth, when the abdomen was opened, small intestine presented, and no colon could be found; for it appeared that the bowel ascended from the left iliac fossa, and making a considerable convolution in the left groin, five inches in length, terminated in the rectum. In a fifth subject the cæcum appeared, and no colon could be reached. The large intestine, on leaving the left iliac fossa, made a convolution upwards, three inches in length, but did not approach the right groin.

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[1] This recommendation of the inguinal operation in certain cases of congenital imperforation does not apply to cases of adults in which colotomy is required to relieve obstruction from disease in the lower bowel. In these cases I prefer, for other reasons, the lumbar operation.

[2] 'Bulletin,' t. xxiv, 1858, 1859, p. 445.

We may infer from these few examinations, that the disposition of the colon in infants, described by Huguier, is not so constant as he states. The colon, at the early period of life, is largely developed, and forms ample convolutions, after reaching the left iliac fossa, varying in extent, however, a good deal in different subjects; and, though its usual course is directed to the right groin, the exceptions are too numerous to render the inguinal operation on the right side as feasible as on the left. But little importance can be attached to the circumstance of an artificial anus on the right side being somewhat nearer the termination of the alimentary canal; and if it seem probable that, after an operation on this side, a prolapsus is less likely to occur from the lower part of the bowel, we might equally expect a greater liability to inversion in the upper portion, which would be free and loose in the abdomen. At present Huguier's suggestion wants altogether the sanction of experience, for I know of no instance in which inguinal colotomy has been performed on the right side.

The following directions for conducting the operative treatment of imperfections of the rectum are based on the results of the preceding inquiry.

1. In simple imperforate anus, the first form, an early attempt should be made to open the bowel in the ano-perineal region. The surgeon should cut down at the site of the anus, exactly in the median line, extending his incision towards the coccyx, dividing the musculo-aponeurotic floor of the pelvis, and penetrating to the depth of an inch and a quarter, and then, if no bowel be reached, the operation should be suspended or abandoned. If no long period have elapsed since birth, and if the infant be not exhausted by sickness or want of nutriment, the surgeon can wait eight or twelve hours, and again examine the wound; for it may happen that the rectum, forced down by the infant's struggles, and finding no resistance from the floor of the pelvis, will project between the borders of the incision.[1] If then any protrusion, or even bulging, be observed, the swelling may be explored with a grooved needle or the point of a bistoury. After the surgeon has reached the bowel, and opened it freely, its coats should be grasped with the forceps, and gently drawn down to the external wound, to the margins of which they should be attached by two or more

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[1] Petit records a case operated on by two surgeons. The first made a conical incision in the perineum, but did not succeed in reaching the rectum. Another surgeon, who saw the infant three hours afterwards, was surprised to find a dark swelling projecting at the wound, and concealing it. This tumour was incised, and meconium escaped. The infant died in seven or eight days, and at the autopsy Petit found that the black tumour was formed by the posterior part of the rectum protruded by the efforts of the infant at the part where the least resistance offered. ('Mémoires de l'Académie Royale de Chirurgie,' t. i, partie ii, p. 240, 1743.)

metallic sutures. The wound must afterwards be poulticed and kept clean, and the sutures may be left in for five days or a week. After a week or ten days a bougie should be passed occasionally, for several weeks at least, even in favorable cases, to obviate any tendency to contraction. When the perineum is rendered convex during the infant's struggles, the operation is pretty sure to be attended with success; but when this part is depressed, and the pelvis obviously small and narrow, the prospect is so unfavorable, that it is questionable whether any attempt should be made to obtain a passage in the anal region. In this case, or in the event of a failure to reach the bowel, the operator should state the circumstances to the parents, and, with their consent, open the colon in the left groin.

2. In the second form, in which an anus opens into a blind sac, an attempt should at once be made to reach the terminal portion of the intestinal canal. The cul-de-sac might, if the symptoms are not too urgent, be first dilated by a sponge tent for a few hours. If the surgeon should feel the upper part of the distended rectum projecting into the lower, he might explore the swelling with a grooved needle, and if gas or meconium escaped, make a free opening with a bistoury. But if nothing be felt to indicate the near proximity of the bowel, he should enlarge the anus by an incision, carried towards the coccyx, so as to divide the posterior wall of the sac, and should pursue his search in this direction to the depth of an inch and a half or two inches from the anus. If the bowel be not reached, this operation should be abandoned, and colotomy performed in the groin. There is seldom any use in delaying the latter operation, in expectation of the bowel becoming more loaded, and descending into the perineal wound, as this malformation is rarely detected until obstruction has already existed for some days. After an opening has been made, if it be obvious, from the depth of the incision, that an interval of some extent exists between the two ends of the bowel, the upper portion must be drawn down and secured to the wound. When a mere septum intervenes, this is not neces-

sary, but careful and even long-continued dilatation will be required afterwards as a security against contraction.

3. In the third form, in which the rectum opens into the urethra, though meconium may escape by the latter passage, the operation should not be delayed, or serious inconvenience may continue, after the establishment of an artificial anus, from previous over-distension of the bowel. The operation should be performed as in the first form, and especial care should be taken that the new channel is made of ample size, in order to afford every facility for the subsequent closure of the communication with the urethra. If the bowel be deeply seated, so as not to be found at a depth of an inch and a half from the surface of the perineum, the operator should desist, and have recourse to colotomy. The difficulty of obtaining the closure of the communication with the urethra renders the treatment of this imperfection the least satisfactory of any of the forms of atresia. After colotomy the entrance into the lower portion of the bowel should be closed with a plug.

4. In the fourth form, in which the rectum opens into the vagina, an immediate operation is not required, but as the abnormal outlet is often insufficient, longer delay than a month should be avoided. A curved director or sound having been passed through the recto-vaginal opening into the bowel, with its point directed to the site of the anus, the surgeon should cut upon it in the median line, being careful to carry his incision backwards towards the coccyx and away from the perineum, in order to preserve as wide a barrier as possible between the abnormal and artificial apertures. The opening into the bowel should be free, and its coats should be secured by sutures to the borders of the external wound. After the artificial anus has been fully established, and all dilatation has ceased to be necessary, steps may be taken to close the recto-vaginal opening. If the aperture be small, its contraction and closure may be effected by touching the edges with the actual cautery; but if it be of large size, a plastic operation will be necessary. The edges may be pared and brought together by metal

sutures, the bowels being kept at rest by opiates for several days, until union is secure.

5. In the fifth form, the abnormal vent being insufficient, an operation is required at an early period. If the opening be situated in the perineum, it may be enlarged by an incision towards the coccyx; but if it be placed in the male as far forward as the scrotum, or in the female close to the vagina, a new passage should be formed at the natural site, a curved director being introduced at the abnormal outlet as a guide to the surgeon in the operation. In both modes of proceeding the mucous membrane of the bowel should be attached by sutures to the skin. Precautionary dilatation will be necessary afterwards.

In the operation of colotomy in the groin in infants, the incision should be made higher and nearer the crest of the ileum than in the adult, and the coats of the colon should be attached by sutures to the margins of the external wound before the bowel is opened, in order to prevent the escape of meconium into the abdominal cavity.

Having in mind the unfavorable issue of the operations performed in the majority of cases of imperfection of the rectum, and the opinions expressed by some authorities against making an artificial anus in such cases, I do not hesitate, nevertheless, in concluding this inquiry, to express my conviction that the surgeon should not shrink from the responsibility of these operations, but should apply what knowledge and skill he can command to the preservation of the infant, and to its protection from future suffering. By careful study, by thoughtful application of the resources of our art, and by increased experience, we may hope to improve the treatment, and to obtain far better results than are recorded here. On what terms life is endurable and worth preserving, may be a subject of anxious consideration to the parents of infants so afflicted; but if it can be shown that in some few cases only these imperfections have been remedied or counteracted, and that life thus saved has been enjoyed and valued, it is surely our duty to persevere, and to strive to overcome the difficulties and obstacles to success.

ON

THE RELATIVE AMOUNTS OF SUGAR AND UREA IN THE URINE

IN

DIABETES MELLITUS.

BY

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The observations were made on two patients, inmates of University College, under the care of Dr. Parkes.

In both cases the urea was estimated quantitatively by Liebig's volumetric method. The usual allowance was made for the chloride of sodium, except in the case of Harriet Meader, in whom it was so small in quantity that no allowance was made. The sugar was estimated quantitatively by the copper test. The water is given in cubic centimètres, and the urea and sugar in grammes.

The following points are attempted to be determined.

1. The relation between the excreted amounts of sugar and urea.

2. The influence of starch and sugar on the excretion of urea.

3. The time at which the sugar reached its maximum after a meal.
4. The duration of the influence of a meal.
5. The temperature of the body.

Case 1.—George Hudson, æt. 30; height, five feet nine inches and a half; weight in health, eleven stone nine pounds. For six months previous to his admission into the hospital he had suffered from symptoms of diabetes, which had increased rapidly in severity, and were strongly marked at the time of his admission. During the first sixteen weeks they continued with unabated activity. After this he improved, and his weight and strength increased. He had some tubercular deposit in both lungs, which had advanced to excavation on the left side. During the early part of the investigation he perspired greatly. During the whole of these observations the patient was purposely kept without medicine. The observations were commenced on October 13th, 1859.

Case 2.—Harriet Meader, æt. 30, of very diminutive stature. She had suffered ten years previously from diabetes, and was greatly emaciated, though she had been in much the same state during the greater part of her illness. She was almost totally blind from double cataract, and her intellect was greatly impaired. She also suffered from a slight leucorrhœal discharge, so that her urine contained a *minute* trace of albumen, which was removed by amylic alcohol before testing for the sugar, but was not removed previous to testing for urea. During her whole illness she was subjected to no treatment, with the exception of having brown bread in the place of white. Nor was any medicine given her whilst these observations were being made.

1. *On the relation between the excreted amount of sugar and urea.*

After the influence of food on the urine had disappeared by long abstinence, a constant ratio was found to be maintained between the sugar and the urea. This ratio was 1 of urea to 2·2 of sugar. The ratio is most strictly observed when several hours are compared together in preference to single hours, as the sugar and urea do not exactly correspond in the exact time of elimination. The following tables show the above ratio clearly:

George Hudson.

Urine in inanition.

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
9th	4·881	2·582	...
10th	4·772	2·415	...
11th	5·350	2·127	...
12th	5·350	2·127	...
13th	4·545	1·598	...
14th	5·025	2·202	...
15th	3·260	1·560	...
Total	33·211	14·612	1 : 2·2

The same ratio was observed on several other occasions; not, as above, in several consecutive hours, but for a single hour only. For, on any day that observations were made, a fasting hour was first taken for comparison. Thus:

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
14th	2·453	1·428	1 : 1·7
14th	5·319	2·430	1 : 2·1
13th	3·977	1·680	1 : 2·3
12th	4·825	1·909	1 : 2·5
			Mean 1 : 2·1

Harriet Meader.

In the following table three consecutive hours are given.

Urine in inanition.

Number of hours since last meal.	Sugar.	Urea.	Ratio of urea to sugar.
15th	1·631	0·727	1 : 2·2
16th	1·631	0·727	1 : 2·2
17th	1·041	0·456	1 : 2·2

But not only was the above ratio observed during inanition hours, but also after a non-amylaceous and non-saccharine diet; for though both the urea and sugar rose greatly, the same ratio was maintained. Here, again, though, when separate hours are compared, the ratio varies slightly, still the mean ratio is the same as that above given.

Hudson.

December 30th.—At 10 a.m. he breakfasted on eggs and mutton. He took tea the evening previous at 5, and had nothing between that time and breakfast.

Urine of nitrogenized food.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
9 to 10 a.m.	4·324	1·676	1 : 2·5
10 to 11 „	3·856	1·583	1 : 2·4
11 to 12 „	3·604	1·972	1 : 1·8
12 to 1 „	3·604	1·972	1 : 1·8
1 to 2 „	4·181	2·618	1 : 1·6
2 to 3 „	8·214	3·832	1 : 2·1
3 to 4 „	5·851	2·970	1 : 1·9
4 to 5 „	4·427	2·380	1 : 1·8
5 to 6 „	3·437	2·042	1 : 1·6
			Mean 1 : 1·97

January 2d.—He dined at 6.30 on fish, mutton, and eggs. He had taken nothing since the evening previous.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
6 to 7 p.m.	0·750	0·583	1 : 1·2
7 to 8 ,,	3·846	2·080	1 : 1·8
8 to 9 ,,	7·937	3·048	1 : 2·6
9 to 10 ,,	9·523	3·744	1 : 2·5
10 to 11 ,,	8·522	3·450	1 : 2·4
11 to 12 ,,	7·283	3·510	1 : 2·0
12 to 9 a.m.	3·945	2·034	1 : 1·9
			Mean 1 : 2·1

The hourly mean, and not the total amount, is given between 12 p.m. and 9 a.m.

4th.—He dined at 11 p.m. on fish and meat.

Hour.	Sugar.	Urea.	Ratio of urea to sugar.
11 to 12 p.m.	8·695	3·520	1 : 2·4
12 to 1 a.m.	6·818	3·300	1 : 2·0
1 to 2 ,,	6·607	3·293	1 : 2·0
2 to 7 ,,	6·666	2·688	1 : 2·4
			Mean 1 : 2·3

Between the hours of 2 and 7 the hourly, and not the total, amount is given.

That some such ratio existed was anticipated on *à priori* grounds, for the sugar, under the above conditions, is probably derived from the nitrogenous elements of the body, and the nitrogen of the albuminous matters which are resolved into sugar must apparently pass off as urea: thus some ratio was probable; for the sugar could only be derived from the nitrogenous or oleaginous constituents of the body. And though reasons have been advanced tending to show that sugar and fat are inter-convertible in these cases, I think it must be admitted that the sugar came from the nitrogenous elements. The fact of the urea maintaining a constant ratio with the sugar in all its variations, under these circumstances, is strong proof of this. For though it may be said that a certain amount of tissue would be consumed in the conversion of fat into sugar; also that urea being the exponent of this, as the tissue used would be in proportion to the sugar formed, so the urea would hold a constant ratio to the sugar; still, the increase of the urea is much too great to be accounted for in this manner, being often double and treble the amount normal to the patient, which is the exponent of all the changes (secretion, &c.) taking place in the body, and would surely more than equal the loss of tissue required to convert so much fat into sugar.

2. *On the influence of amylaceous or saccharine food on the excretion of urea.*

The excretion of urea appears to be increased after amylaceous or saccharine food, and this independent of the kind of sugar used. The following tables render this probable.

HARRIET MEADER.

December 17th.—The patient dined at 4 on mixed diet. At 12 p.m. eight drachms of cane sugar were given to her.

Hour.	Quantity.	Sugar.	Urea.
9 p.m.	261 c.c.	14·085	1·357
12 "	220 "	11·000	1·166
1 a.m.	235 "	13·976	1·222
2 "	282 "	14·100	1·381
3 "	225 "	10·416	1·125
9 "	140 "	7·954	0·840

The above (as is the case with the following) experiment is not so perfect as could have been desired. As it was impossible to keep her without food till the influence of the previous meal had disappeared. Still, the sugar was never given her till the urinary sugar and urea had commenced to decline.

In the above table it is seen that the urea was increased in the second hour after the sugar was taken by 0·220 gramme. The actual increase was much more than this; for the urea would have continued to decrease in amount during these two hours if no sugar had been given, the influence of the previous meal during that time becoming less.

19th.—The patient had taken no food since 11 p.m. the evening previous; at 10 a.m. eight drachms of sugar of milk were given to her.

Hour.	Amount.	Sugar.	Urea.
8 to 9 a.m.	238 c.c.	11·053	1·528
9 to 10 „	107 „	6·079	0·920
10 to 11 „	150 „	8·333	1·120
11 to 12	165 „	9·375	1·270
12 to 1 p.m.	102 „	6·071	0·683

19th and 20th.—She dined heartily at 4 p.m. on mixed diet. At 12 p.m. she had eight drachms of sugar of milk given her.

Hour.	Amount.	Sugar.	Urea.
9 to 10 p.m.	101 c.c.	5·295	0·671
10 to 12	195 „	9·461	1·482
12 to 1 a.m.	255 „	11·590	1·785
1 to 2 „	230 „	10·865	1·610
2 to 3 „	158 „	8·977	1·216

20th and 21st.—The patient dined, as usual, on a mixed diet at 4 p.m., and at 12 p.m. eight drachms of sugar of milk were given to her.

Hour.	Amount.	Sugar.	Urea.
7 to 8 p.m.	305 c.c.	16·576	1·830
8 to 10 „	250 „	12·019	1·550
10 to 12	187 „	10·190	1·325
12 to 1 a.m.	210 „	10·744	1·449
1 to 2 „	234 „	10·833	1·544
2 to 3 „	195 „	9·375	1·267

22d.—She breakfasted at 9 on mixed diet. At 1 p.m. eight drachms of glucose were given her.

Hour.	Amount.	Sugar.	Urea.
10 to 11 a.m.	286 c.c.	16·388	1·510
11 to 12	258 „	13·913	1·522
12 to 1 p.m.	55 „	3·197	0·368
1 to 2 „	195 „	11·079	1·092
2 to 3 „	187 „	12·331	0·990
3 to 4 „	130 „	9·027	0·728

On the same day she dined, as usual, at 4 p.m. At 12 p.m. she had eight drachms of glucose given her.

Hour.	Amount.	Sugar.	Urea.
8 to 9 p.m.	243 c.c.	13·522	1·849
9 to 12	253 „	11·727	1·570
12 to 1 a.m.	280 „	12·096	1·820
1 to 3 „	...	5·062	1·582
3 to 9 „	...	6·713	1·203

GEORGE HUDSON.

January 5th.—The evening previous he took a non-amylaceous dinner; consequently the sugar and the urea had nearly reached the ratio observed during inanition hours, and the influence of the meal was much more completely worked off than in the previous tables, thus more correctly showing the actual increase after sugar was taken. Four ounces of cane sugar were given him at 9 a.m.

Hour.	Amount.	Sugar.	Urea.
8 to 9 a.m.	60 c.c.	4·166	1·600
9 to 10 „	126 „	9·834	2·102
10 to 11 „	142 „	12·241	2·101
11 to 12	127 „	11·759	1·778
12 to 1 p.m.	82 „	8·200	0·984

In all the above tables, when more than an hour elapsed before the urine was examined, the total amount of water, sugar, and urea is not given, but the mean hourly amount.[1]

This fact is also well shown on other occasions, in other experiments not devised for this special purpose. Thus, after a hearty non-amylaceous meal, the urea was found to be much less than after a mixed diet, though it may be fairly granted that the amount of nitrogenized food taken was greater in the former than the latter kind of meal. Thus, on December 30th, he took a non-amylaceous breakfast; the highest hourly amount of urea was 3·832 grammes (59 grains). On January 2d he had a similar dinner, and the highest hourly amount of urea was 3·744 (58 grains). On both these occasions he ate most heartily. On October 24th, after a mixed breakfast, the highest hourly amount of urea was 5·3 grammes (82 grains). After a similar tea on the same day, 9·2 grammes (142 grains). On October 28th, after a mixed breakfast, the urea reached 5·355 grammes (83 grains). On the same day, after a similar tea, 7·7 grammes (119 grains) per hour. These are enough for examples, though many others might be given.

The facts above attempted to be established—namely, that during inanition, and also after a meal containing nitrogenous matter, whether combined with amylaceous matter or not, albuminous matter is converted into sugar and urea, and that this is greatly increased by the administration of sugar, and therefore by an amylaceous diet—are quite sufficient to account for the great loss of weight and

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[1] In the following case, a patient, æt. 11, under the care of Dr. Hare, half a pound of sugar was given at 12 p.m., the urine having been collected for the two hours previous, and again from 12 p.m. to 7 a.m.

Hour.	Sugar.	Urea.
10 to 12 p.m.	4·049	1·322
12 p.m. to 7 a.m.........	12·668	3·230

As usual, the hourly amount is given in the above table.

During the above experiment, the patient took no food between 5 p.m. and 8 a.m. next morning.

In the above table the hourly amounts of sugar and urea are given.

strength in these patients, and the general degeneration of the functions of the body.

It may, perhaps, be said that the increase of the urea, after the administration of sugar, was due to this causing an increase in the amount of urinary water, under which circumstances it is known that an increase of urea takes place.[1] But the increase then depends on previous retention, probably due to a deficiency in the amount of urinary water. Now, it cannot be said that in diabetic patients there is any deficiency in this.

Again, if the increase in the amount of urinary water were the cause of the increase in the amount of urea, there should be a relationship between the two, but nothing of the kind holds, as is seen from the following table, in which the water and urea are given for the twenty-four hours, and the ratio between them also. On all these days the patient took ordinary mixed diet.

HUDSON.

Date.	Water.	Urea.	Ratio of urea to water.
October 17th	5575 c.c.	82·339	1 : 68
18th	5354 „	83·037	1 : 64
19th	5129 „	74·258	1 : 69
February 14th	3805 „	33·454	1 : 115
15th	4042 „	32·336	1 : 126
16th	4400 „	30·800	1 : 146
18th	4000 „	25·600	1 : 160

The first three closely correspond; but, as the water is obviously under the control of the sugar and the urea also, when there is a ratio between the two latter there will of necessity be a relation between the water and urea, and this occurred on the first three days given.

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[1] Thus, in a healthy subject, of average height and build, by drinking a pint of water hourly, the amount of urea rose from 2·0 grms. per hour to 4·040 grms. for three hours. During the fourth hour, however, it fell to 3·026. It is true that the usual allowance was not made for the NaCl; but the amount of urinary water was increased enormously, whilst the NaCl suffered no such increase.

The above table, I think, shows indubitably that the increase in the urea is independent of the increase of urinary water, as the ratio between the two varies from 1 : 68 to 1 : 160.

On referring to this table, showing the increase in the amount of urea and sugar after a non-saccharine and non-amylaceous meal, it will be seen that an enormous increase in the amount of the urea resulted. It has also been shown that there is probably an increased formation of urea in the body, due to the ingestion of saccharine matter; but the amount of urea eliminated by one or both of these causes may vary in different patients, and in the same patient in different periods of the disease. From the above cases it appears that the condition of the patient depends on the amount of urea, and not on the amount of sugar, eliminated; that, in fact, the improvement of the patient is due to a diminished amount of urea formed, whilst, at the same time, the amount of sugar eliminated may remain the same or be greatly increased.[1] Hudson's case illustrates this. At first he lost flesh with great rapidity, all his symptoms being proportionately severe. The disease continued with unabated severity for nine months. He then apparently remained in *statu quo* for a few weeks, but towards the latter part of the investigation he improved greatly in strength and weight. These variations are well seen in the fluctuations in his weight. Thus, in health he weighed 163 lbs.; January 3d, 107 lbs.; January 26th, 103 lbs.; February 19th, 113 lbs.; February 25th, 113 lbs.

On referring to the following table, it will be seen that coincident with this improvement was an increase in the amount of excreted sugar (this increase was accounted for by an increase in his appetite). But at the same time that this increase in the sugar occurred, a large diminution in the amount of urea took place. Thus, during October, November, and December, he rapidly declined; but in January and February he improved greatly.

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[1] Of course, in judging of the increase of the urea in these cases, the amount of nitrogenous food ingested must be considered.

Date.	Sugar.	Urea.
October 17th	324·840	82·339
18th	342·413	83·037
19th	313·091	74·258
January 26th	228·947	29·928
27th	339·285	34·960
28th	231·761	32·637
30th	366·935	41·860
31st	340·357	40·026
February 1st	389·224	30·702
2d	327·307	19·743
3d	281·774	33·542
4th	562·500	42·120
6th	437·900	28·901
7th	420·600	41·639
8th	446·354	44·564
9th	512·000	29·696
10th	456·250	42·705
11th	609·659	40·237
13th	541·666	32·240
14th	413·586	33·454
15th	459·318	32·336
16th	392·857	30·800
18th	313·500	25·600

That the severity of the symptoms is greatly due to the urea is also rendered probable from three other cases, one of which is given above, the other two Dr. Garrod kindly allowed me to take. In all these the amount of sugar excreted was nearly as large as the amount in the case of Hudson during the most severe part of his disease (October), but the amount of urea was very much less, and at the same time their condition was stationary, and had been so over a period of about ten years.

The following table shows the daily amount of sugar and urea in the case of Harriet Meader, on mixed diet.

Date.	Sugar.	Urea.
December 16th	189·435	23·300
17th	248·504	26·098
18th	238·712	33·653
19th	257·475	30·196
23d	247·589	33·885

Dr. Garrod's cases were taken for only one day each; they gave the following amount of sugar and urea:

	Sugar.	Urea.
1st..............	295·454	39·000
2d..............	264·309	32·374

The variation in the amount of urea in different patients, and in the same patient at different periods of the disease, may be due either to a lessened influence of the ingested sugar or to a diminished amount of nitrogenous matter resolved into sugar and urea, independent of the influence of sugar (which we have seen occurs), or, as is probable, it may be due to both causes combined.

This twofold origin of urea will readily account for their enormous appetite; for the chief part of the albuminous matter ingested being resolved into urea and sugar, proportionately less will be left to nourish the tissues, and if the entire amount of nitrogenous matter should be eliminated, then we can understand how it is that they experience a sense of hunger immediately after a meal. And if it be established that the ingestion of sugar has the power to increase the resolution of albuminous matters into urea and sugar, it is conceivable that the amount thus resolved may be even greater than the amount taken in as food; then the blood or other tissue will be attacked, and the meal will then leave the patient in a worse condition than it found him.

These facts also explain how it is that abstinence from amylaceous matter, as food, is often followed by such good results.

The urea in Hudson's case increased after a meal, corresponding, in this respect, to the increase that occurs in health, except as regards amount; and there can be little doubt, I think, that the increase in his case was due to the same causes that operate in health. But as the sugar increased also in his case, maintaining the ratio mentioned above, after a non-amylaceous meal, if it can be shown from whence the sugar is derived it will also show the origin of this rise in the urea.

Now, as regards the increase of the sugar, there are two conceivable sources—1st, the tissues (retrograde metamorphosis); 2d, some organ, by its function, resolving nitrogenous matter into sugar and urea, which function, in diabetes, is altered, so that the sugar is less highly elaborated, and thus non-consumed.

Again, the sugar during inanition periods, and the increase after a non-amylaceous meal, are probably derived from the same source.

The chief difficulty in settling this question lies in the difficulty of proving whether any of the sugar, either taken in *ab externo*, or formed in the body, is consumed, or whether all is thrown off (as far as these cases are concerned).

There are reasons for thinking that some is consumed; thus, the enormous rise in the temperature after a meal would seem to point to this conclusion, though, as the diet in all these cases was mixed, it might have been due to oleaginous matters, taken in in unusually large amount. On the other hand, there are some rather strong reasons for thinking that all the sugar was thrown off. For, though the temperature rose so highly after food, this did not stand in proportion to the amount of amylaceous food taken; thus the temperature reached its highest after dinner, when the nitrogenous and oleaginous elements were in excess, the patient then taking much less bread. On October 24th, after dinner, it reached 101°; the same evening the temperature rose nearly as high as $100\frac{2}{5}^\circ$, but he had a meat tea; he ate, however, much more bread than at dinner. On October 27th he had a very good breakfast, but only toast and tea; the temperature reached $99\frac{3}{5}^\circ$. And on the 28th, after a very good tea, it reached only $99\frac{4}{5}^\circ$, and it must be remembered that he took butter with his bread at breakfast and tea, so that would, perhaps, account for the great rise, even then.

Again, as there is a constant ratio between the sugar and the urea during inanition and after a non-amylaceous diet, it is obvious that if any is consumed it must be complete up to a certain point, and after that in a constant ratio; if this applies to sugars formed in the body, it probably applies to all. If so, it is evident that there should be a relationship between the sugar excreted and the temperature (if the former is the cause of the rise of the latter); or if the temperature is due to the sugar consumed, and this again holds a constant proportion to that excreted, the excreted amount will hold a proportion to the temperature; but there is not the slightest relationship between the two. It may be said that both the sugar and the oleaginous matters were concerned in the elevation of the temperature, and as the proportion of the two to one another, in the food, varied, so no relationship would exist between the sugar and the temperature. But admitting the force of this, still, if any were due to the sugar, there could not be so great a discrepancy as constantly exists between the temperature and the sugar, no vestige of a relationship existing between them. This is well seen by looking at the diagrams, where it will be observed that, on October 24th, at 3 p.m., the temperature stood at 101°, whilst the sugar amounted to 17 grammes per hour. At 10 p.m. the temperature stood at $100\frac{2}{5}$° ; the sugar had reached 37 grammes per hour.

27th.—The temperature at 11 a.m. was $99\frac{2}{5}$°, and the sugar stood at 30 grammes.

28th.—At 9 p.m. the temperature showed 99°. The sugar reached 31 grammes per hour.

It thus appears to me that the temperature cannot be taken to prove that any of the sugar is consumed, indeed, the inference is in the other direction.[1]

It may be that some of the sugar taken in *ab externo* was consumed, whilst all that formed in the body was excreted. If this be granted, it is all that is required for the present purpose.

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[1] There is, however, another conceivable cause of the rise in the temperature. Thus it is known that at least some of the sugar ingested is converted into lactic acid in the alimentary canal, and being absorbed as such would be consumed, and cause an elevation in the temperature. (See Lehmann's 'Physiological Chemistry,' translated by the Cavendish Society.)

When abstinence of food was continued long enough, in both cases the ratio between the sugar and the urea was lost, that is to say, the urea ceased to fall, whilst the sugar still did so. Thus, in Hudson's case, on January 2d, the following occurred:

Hour.	Sugar.	Urea.
1·30	2·884	1·320
3	1·384	1·100
5	0·635	1·000
7	0·750	0·583

MEADER.

1·30	1·291	0·574
2·30	0·852	0·567
3·30	0·961	0·700

Now, if no sugar is consumed, to what is the excess, just at this point due? If the sugar is due to some organ acting on the albuminous constituent of the blood, and not due to the retrograde metamorphosis of the tissues, we should have a small amount of urea to be accounted for in this manner; and whilst the sugar and urea are very large in quantity, this would be insufficient to affect the ratio to any appreciable degree; but when the sugar and urea become much reduced, then the urea would be found in excess, as we find it here. For if the sugar is derived from the retrograde metamorphosis of the tissues, then they should fall, *pari passu,* maintaining the usual ratio. Thus I think we are driven to admit that the sugar is due, in these cases, to some organ which is endowed with this function; and even if some of the sugar was derived from the tissues by retrograde metamorphosis, the liver must have been at fault likewise. As it is known that it is here that ordinary sugar is rendered available for the body, and as it is also known that the liver has the power of forming sugar from nitrogenous substances, it is more compatible with reason to ascribe the entire disease to the liver than to distribute it between it and the rest of the body.[1]

Thus, having shown it probable that the sugar in these cases was due to the function of the liver, it is of course in an equal degree probable that the rise of urea after a meal is due to the same organ.

From what has gone before in this paper, I think it appears that the excretion of urea from the influence of amylaceous food, and the excretion due to nitrogenous food, though mostly combined, are not necessarily so; that the sugar taken in may pass off unchanged, whilst the nitrogenous matters remain unattacked; but whether the reverse may occur is, perhaps, doubtful,[2] though conceivable. But besides these two possible forms of diabetes, there is probably a third, namely, the tissues which by altered retrograde metamorphosis, yield diabetic sugar and urea instead of their ordinary products.

Any theoretical consideration as to the nature of the lesion (in cases similar to those given above), based solely on the non-consumption of the sugar, if not erroneous, must be incomplete, as it is necessary to consider the excessive formation of sugar in the body. Thus we may reject as at least insufficient Miahle's view, that the disease is due to an insufficient amount of alkali in the blood; also any views that look to the respiratory organs as at fault; as also any view that merely considers the disease due to mal-assimilation of sugar in the intestinal canal; and further, any system of treatment founded on such views must be received with caution.

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[1] Again, the liver (as is well seen in carnivora) forms sugar at the expense of the albuminous constituents of the body, the nitrogen of which must pass off as urea; and as we find an increased formation of sugar by the liver after a meal, so should we expect a corresponding increase in the amount of urea in the urine. Dr. Parkes, moreover, has endeavoured to show that in cases of abscess of the liver, with great loss of liver-tissue, the amount of urea is diminished. Another probable cause of part of the increase is the products of retrograde metamorphosis taken in with muscle, &c., as food, for we know the juice of flesh contains creatine and creatinine, &c. These would probably be absorbed to some extent, and some would pass on to the formation of urea.

[2] Since the above was written I have met with three cases which render it possible that such may be the case.

3. *On the time at which the sugar reached its maximum in the urine after a meal.*

This varies according to the nature of the food and the stage of the disease.

Mixed diet:

Hudson.

During the early experiments it reached its maximum during the third or fourth hour.

Date.	Hour.		
	Breakfast.	Dinner.	Tea.
October 17th	3d	—	—
18th	4th	2d	—
21st	—	—	3d
24th	—	—	4th
26th	3d	—	—
27th	4th	—	—
28th	4th	—	—

Later in the disease it reached its highest during the sixth hour.

January 6th	6th.
January 13th	6th.

Sugar in solution (both cases) reached its maximum at the second hour, with one exception, when the maximum was reached during the first hour.

In these observations the time occupied in eating the meal is included.

Pure albuminous diet:

Date.	Hour.		
	Breakfast.	Dinner.	Tea.
November 2d..................	—	4th	—
December 29th	—	4th	—
January 2d	—	4th	—

4. *Duration of the influence of food* (*mixed diet*).

The duration of the influence of mixed diet was longer towards the latter part of the observations.

October 19th.—Tea between 5 and 6 p.m. All effect of the meal had disappeared at 2 next morning, that is to say, the starvation ratio was reached by that time; but on December 28th the influence still existed after thirteen hours had elapsed, and might have continued longer, but the patient could wait no longer. In this woman the influence lasted fifteen hours. Thus it would appear, as might have been expected, that the digestive powers became impaired.

The urea had a tendency to pass off sooner somewhat than the sugar, possibly because it is more pernicious.

The increase of the sugar in the urine due to the influence of the meal lasted longer than the increase in the urea.

The above statements will be found to be correct on referring to the tables appended, in which the entire observations are given.[1]

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[1] Besides the sugar and urea, the amount of urinary water in all cases is given; also in many cases the chloride of sodium, and in a few cases the specific gravity. No reference has been made in the previous pages to these constituents, as the observations were not sufficiently numerous.

Hudson.

Till October 20th he was allowed to take what food he chose. This consisted of bread and butter, tea or cocoa, with sugar and milk, for breakfast. Meat, bread, and potatoes, with half a pint of porter, for dinner. His tea corresponded to his breakfast.

October 14th.—Breakfasted at 8·30; dined at 1; tea at 5.

15th.—Breakfasted at 9; dined at 1; tea at 5.

16th.—Breakfasted at 9·15; dined at 1; tea at 6.

17th.—Breakfasted at 8·30; dined at 1.

18th.—Breakfasted at 9; dined at 1; tea at 6.

19th.—Breakfasted at 9; dined at 1; tea at 5·30.

20th.—Breakfasted at 8.

OCTOBER 13TH.

Hour.	Quantity.	Sugar.	Urea.	NaCl.	Sp. gr.
9 to 10 p.m.	390 c.c.	...	4·524	...	1012
10 to 11 „	355 „	...	4·473	...	1042
OCTOBER 14TH.					
11 p.m. to 8 a.m.	1115 c.c.	...	23·192	6·244	1050
8 to 9 a.m.	60 „	...	1·710	0·300	
9 to 10 „	205 „	...	3·895	0·820	
10 to 11 „	310 „	...	4·030	1·054	
11 to 12	330 „	...	2·640	1·386	
12 to 1 p.m.	295 „	...	2·006	0·914	
1 to 2 „	367 „	...	3·596	1·388	
2 to 3 „	335 „	...	3·886	1·239	
3 to 4 „	247 „	...	3·507	0·666	
4 to 6 „	377 „	...	7·351	1·508	
OCTOBER 15TH.					
6 p.m. to 8 a.m.	2600 c.c.	...	36·920	8·840	
8 to 9 a.m.	50 „	...	1·300	0·185	
9 to 10 „	80 „	...	2·136	0·320	
10 to 11 „	150 „	...	2·775	0·330	
11 to 12 „	265 „	...	3·445	0·530	
12 to 1 p.m.	265 „	...	3·445	lost	
1 to 2 „	333 „	...	3·996	1·198	1042
2 to 3 „	315 „	...	4·095	1·102	1043
3 to 4 „	265 „	...	4·081	0·715	1047
4 to 5 „	180 „	...	3·366	0·480	1051
5 to 6 „	225 „	...	4·320	0·832	1048
6 to 7 „	280 „	...	4·844	1·164	1047
7 to 8 „	350 „	...	4·865	1·120	1043
8 to 9 „	275 „	...	3·575	0·825	1045
OCTOBER 16TH.					
9 p.m. to 8 a.m.	1480 c.c.	...	23·680	6·600	
8 to 9 a.m.	50 „	...	1·270	0·225	
9 to 10 „	88 „	...	2·041	0·440	
10 to 11 „	220 „	12·200	3·580	0·572	
11 to 12 „	290 „	16·111	3·915	0·870	
12 to 1 p.m.	295 „	17·987	3·953	1·357	
1 to 2 „	360 „	25·714	4·080	1·476	
2 to 3 „	250 „	15·625	3·025	1·075	
3 to 4 „	397 „	20·253	5·161	3·295	1041
4 to 5 „	215 „	15·808	3·289	1·258	1050
5 to 6 „	215 „	14·527	3·504	1·225	1051
OCTOBER 17TH.					
6 p.m. to 8 a.m.	2225 c.c.	163·603	36·295	17·132	1049
8 to 9 a.m.	127 „	7·434	2·870	1·143	1051
9 to 10 „	225 „	14·423	3·487	1·755	1045
10 to 11 „	265 „	16·562	2·982	2·252	1040
11 to 12 „	590 „	26·339	6·608	3·304	1040

OCTOBER 17TH—*continued.*

Hour.	Quantity.	Sugar.	Urea.	NaCl.	Sp. gr.
12 to 1 p.m.	515 c.c.	23·842	6·025	3·399	1038
1 to 2 „	400 „	18·181	4·880	2·600	1036
2 to 3 „	465 „	16·146	5·673	3·441	1035
3 to 4 „	330 „	16·176	4·851	1·710	1040
4 to 5 „	215 „	13·109	4·178	0·838	1045
5 to 6 „	215 „	9·045	4·493	1·075	1050
OCTOBER 18TH.					
6 p.m. to 8 a.m.	2535 c.c.	147·384	43·095	9·126	1046
8 to 9 a.m.	52 „	2·453	1·428	0·273	1050
9 to 10 „	172 „	12·647	3·698	0·584	1045
10 to 11 „	255 „	16·346	3·493	0·994	1039
11 to 12 „	380 „	25·000	4·408	1·444	1040
12 to 1 p.m.	407 „	28·208	4·354	1·465	1039
1 to 2 „	443 „	24·611	4·562	1·993	1042
2 to 3 „	400 „	33·333	4·520	1·200	1045
3 to 4 „	270 „	15·000	3·591	1·215	1044
4 to 5 „	325 „	18·460	5·037	1·430	1040
5 to 6 „	315 „	13·815	4·851	1·795	1040
6 to 7 „	195 „	10·156	3·315	0·936	1048
7 to 8 „	270 „	21·094	3·051	0·864	1047
8 to 9 „	327 „	26·435	4·152	1·113	1046
OCTOBER 19TH.					
9 p.m. to 8 a.m.	1510 c.c.	92·037	26·275	6·946	1049
8 to 9 a.m.	100 „	5·319	2·430	0·750	1050
9 to 10 „	95 „	5·525	2·004	0·351	1050
10 to 11 „	237 „	17·426	3·578	0·616	1045
11 to 12 „	327 „	18·055	4·087	1·012	1042
12 to 1 p.m.	345 „	19·166	3·795	1·301	1040
1 to 2 „	388 „	22·558	4·362	1·590	
2 to 3 „	440 „	22·000	5·325	2·288	
3 to 4 „	395 „	21·944	4·891	2·409	
4 to 5 „	290 „	16·122	3·944	1·524	
5 to 6 „	250 „	15·244	4·050	1·175	
6 to 7 „	228 „	17·272	3·898	0·661	
7 to 8 „	313 „	22·357	4·695	0·939	
8 to 9 „	380 „	29·688	4·332	1·140	
9 to 10 „	435 „	21·750	5·176	1·696	
10 to 11 „	383 „	21·277	4·251	1·532	
11 to 12 „	342 „	21·625	4·856	1·675	
OCTOBER 20TH.					
12 to 1 a.m.	248 c.c.	12·917	2·429	1·288	
1 to 2 „	207 „	11·500	2·607	1·304	
2 to 3 „	123 „	4·881	2·583	1·210	
3 to 4 „	105 „	4·772	2·415	0·987	
4 to 6 „	197 „	10·701	4·255	1·635	
6 to 7 „	72 „	4·545	1·598	0·597	
7 to 8 „	97 „	5·052	2·201	0·834	
8 to 9 „	60 „	3·260	1·560	0·510	

For the tables on Oct. 26th, 27th, and 28th, the reader is referred to the diagrams.

December 28th.—Dined between 12 and 1; ordinary diet. Tea at 5·15; ordinary diet. At 6·45 he took some milk.

29th.—6 a.m., he took a non-amylaceous breakfast. Tea at 5.

30th.—Breakfasted between 9 and 10, on a non-amylaceous meal. He took nothing from that time till 6, when he dined on a mixed diet.

31st.—He took only breakfast and dinner, the latter at 6 p.m.

January 2d.—Breakfasted at 9, on a non-amylaceous diet; but scarcely ate anything till 6 p.m., when he ate a hearty dinner, composed of non-amylaceous matter.

4th.—He took nothing from breakfast till 6 p.m., when he took a pint of milk. At 11 p.m. he ate heartily of meat alone.

5th.—9 a.m., he took two ounces of cane sugar.

DECEMBER 28TH.

Hour.	Quantity.	Sugar.	Urea.
4 to 5 p.m.	308 c.c.	22·000	4·312
5 to 6 "	312 "	22·941	4·368
6 to 7 "	510 "	36·428	5·304
7 to 8 "	305 "	22·426	3·233
8 to 9 "	252 "	20·321	2·973
9 to 10 "	394 "	30 781	4·275
DECEMBER 29TH.			
10 p.m. to 5 a.m.	1860 c.c.	122·368	26·412
5 to 6 a.m.	122 "	8·714	2·293
6 to 7 "	107 "	7·037	2·204
7 to 8 "	105 "	6·907	2·268
8 to 9 "	98 "	6·447	2·254
9 to 6 p.m.	2160 "	154·285	35·424
6 to 7 "	363 "	23·881	5·445
7 to 8 "	345 "	20·535	5·106
8 to 9 "	190 "	11·046	3·268
9 to 12·30 a.m.	567 "	35·437	11·907
DECEMBER 30TH.			
12·30 to 8 a.m.	580 c.c.	45·312	13·456
8 to 9 a.m.	62 "	5·000	1·475
9 to 10 "	64 "	4·324	1·676
10 to 11 "	57 "	3·856	1·503

December 30th—*continued.*

Hour.	Quantity.	Sugar.	Urea.
11 a.m. to 1 p.m.	124 c.c.	7·208	3·944
1 to 2 p.m.	77 „	4·181	2·618
2 to 3 „	138 „	8·214	3·832
3 to 4 „	110 „	5·815	2·970
4 to 5 „	85 „	4·427	2·380
5 to 6 „	66 „	3·437	2·042
January 2d.			
9 p.m. to 7·30 a.m.	2320 c.c.	45·238	26·216
7·30 to 8·30 a.m.	78 „	4·642	1·255
8·30 to 9·30 „	124 „	9·687	1·996
9·30 to 10·30 „	120 „	8·571	1·920
10·30 to 11·30 „	110 „	7·432	1·980
11·30 to 12·30 p.m.	87 „	5·178	1·705
12·30 to 1·30 „	60 „	2·884	1·320
1·30 to 3 p.m.	64 „	2·077	1·651
3 to 5 p.m.	61 „	1·270	2·000
5 to 7 „	72 „	1·500	2·332
7 to 8 „	80 „	3·846	2·080
8 to 9 „	127 „	7·937	3·048
9 to 20 „	160 „	9·523	3·744
10 to 11 „	150 „	8·522	3·450
11 to 12	134 „	7·283	3·510
12 to 9 a.m.	625 „	35·511	18·312
January 4th.			
7·30 to 8·30 p.m.	503 c.c.	26·198	9·355
8·30 to 11 p.m.	500 „	22·727	8·950
11 to 12	160 „	8·695	3·520
January 5th.			
12 to 1 a.m.	150 c.c.	6·818	3·300
1 to 2 „	185 „	6·607	3·293
2 to 7 „	640 „	33·333	13·440
7 to 8 „	153 „	10·066	3·457
8 to 9 „	60 „	4·166	1·600
9 to 10 „	126 „	9·834	2·102
10 to 11 „	142 „	12·241	2·101
11 to 12	127 „	11·759	1·778
12 to 1 p.m.	82 „	8·200	0·984

MEADER.

On December 15th, the urine was saved for the entire day, having been kept also through the night previous, from 12·45 a.m. At 5 p.m. of this day the patient ate a hearty tea; from this time she took no food till 11 a.m. December 16th, when we ceased collecting the urine for a few hours.

The same evening (December 16th) the patient ate a bad tea, at 5 p.m., composed of ordinary diet. The urine was collected from 8 p.m. The patient took no food till 12 a.m. next morning (December 17th). Thus she had no food for 18 hours. At 4 p.m. she took dinner and tea together. The urine was now continuously collected (from 8 p.m.); sometimes hourly; at others, during various periods, to 3 a.m. December 21st. December 17th, at 12 p.m., the patient took eight drachms of cane sugar, in 275 c.c. of water. At tea, the previous evening, she drank 570 c.c., and another 180 c.c. at 1 a.m.; beside this, she drank nothing till breakfast, at 9 a.m. the following morning.

December 18th.—At 11 p.m., took a mug of tea and a round and a half of toast.

19th.—At 10 a.m., took eight drachms of sugar of milk in 275 c.c. of water, the patient having taken no food since the tea and toast on the previous evening. On the same day she dined at 4 p.m., on fish, mutton chop, bread, and tea; during the rest of the evening she took nothing but water. At 12 p.m., eight drachms of sugar of milk, in the usual quantity of water, was given her.

20th.—At 3 a.m., she took some bread and butter and tea. Breakfasted at 10. Dined at 4, on a similar diet to that of the day before. 12 p.m., she drank eight drachms of sugar of milk, in 275 c.c. of water.

DECEMBER 14TH.			
Hour.	Quantity.	Sugar.	Urea.
5 to 9·15 p.m.	125 c.c.	2·976	1·975
9·15 to 10·45 p.m	83 ,,	1·921	1·045
10·45 to 11·45 ,,	155 ,,	3·799	1·813
11·45 to 12·45 a.m.	117 ,,	3·052	1·240
DECEMBER 15TH.			
12·45 to 9 a.m.	2090 c.c.	96·759	15·048
9 to 2 p.m.	1205 ,,	37·631	7·230
2 to 12 ,,	1890 ,,	98·437	11·340
DECEMBER 16TH.			
12 to 9 a.m.	1025 c.c.	53·385	6·563
9 to 11 ,,	44 ,,	2·291	1·410

Hour.	Total Quantity.	Total Sugar.	Total Urea.
December 16th and 17th.			
8 to 10 p.m.	503 c.c.	25·150	4·024
10 to 11 „	272 „	12·362	2·067
11 to 1 a.m.	345 „	16·273	3·036
1 to 2 „	210 „	4·687	1·680
2 to 9 „	635 „	23·694	6·350
9 to 11 „	107 „	3·262	1·458
11 to 12 „	30 „	1·041	0·456
12 to 4 p.m.	570 „	29·688	4·104
4 to 9 „	1302 „	70·924	6·786
9 to 12 „	660 „	33·000	3·496
December 18th.			
12 to 1 a.m.	235 c.c.	13·976	1·222
1 to 2 „	282 „	14·100	1·381
2 to 3 „	225 „	10·416	1·125
3 to 9 „	840 „	47·727	5·040
9 to 11 „	217 „	12·616	2·039
11 to 3·30 p.m.	680 „	35·836	3·400
3·30 to 7 p.m.	1200 „	60·000	5·520
7 to 9 p.m.	375 „	20·833	2·875
December 19th.			
9 p.m. to 9 a.m.	2865 c.c.	132·638	18·336
9 to 10 a.m.	107 „	6·079	0·920
10 to 11 „	150 „	8·333	1·120
11 to 12 „	165 „	9·375	1·270
12 to 1 p.m.	102 „	6·071	0·683
1 to 4 „	1035 „	49·759	7·969
4 to 10 „	610 „	31·770	4·026
10 to 12 „	390 „	18·923	2·964
December 20th.			
12 to 1 a.m.	255 c.c.	11·590	1·785
1 to 2 „	230 „	10·865	1·610
2 to 3 „	158 „	8·977	1·216
3 to 5 p.m.	2873 „	149·719	16·683
5 to 7 „	375 „	23·439	1·987
7 to 8 „	305 „	16·576	1·830
8 to 10 „	500 „	24·038	3·100
10 to 12 „	375 „	20·380	2·625
December 21st.			
12 to 1 a.m.	210 c.c.	10·744	1·449
1 to 2 „	234 „	10·833	1·544
2 to 3 „	195 „	9·375	1·267

An intermission then occurred, and the urine was again collected from 9 a.m., December 22d, and kept from this time till 1 p.m., December 24th, over various periods.

December 22d.—She breakfasted at 9; breakfast consisted of bread and butter, with cocoa, with milk and sugar. At 1 p.m. she took eight drachms of glucose, in 275 c.c. of water. Dinner between 4 and 5, consisted of usual food. At 12 p.m. took another eight drachms of glucose, in same amount of water.

23d.—Breakfasted at 9; had three eggs, and tea without milk or sugar, thus solely albuminous food. Dined at 2, good; usual food. Tea at 5; bread and butter; then had nothing till next morning.

24th.—At 9 a.m. drank a mugful of tea; another at 10·20, and then had four eggs. The tea, on both occasions, was without milk or sugar. Dined at 2; usual food.

25th.—Took no starch food on this day, from 6 p.m. (nor milk).

26th.—Breakfasted, at 9·30, on egg and tea, without milk or sugar. Between 1 and 2, had meat solely. At 5 p.m. drank a solution containing three drachms of glucose. At 9 p.m. had meat and a very small piece of bread.

27th.—Breakfasted on eggs and tea, without sugar or milk, at 9·30. Dined, at 1·45, on chop and beef tea solely.

DECEMBER 22D.

Hour.	Quantity.	Sugar.	Urea.
9 to 11 a.m.	472 c.c.	32·777	3·020
11 to 12	258 "	13·913	1·522
12 to 1 p.m.	55 "	3·197	0·368
1 to 2 "	105 "	11·079	1·092
2 to 3 "	187 "	12·331	0·991
3 to 4 "	130 "	9·027	0·728
4 to 9 "	1217 "	67·611	9·249
9 to 12	760 "	35·183	4·712
DECEMBER 23D.			
12 to 1 a.m.	280 c.c.	12·069	1·820
1 to 3 "	255 "	10·125	3·162
3 to 9 "	145 "	40·277	7·221
9 to 10 "	54 "	2·596	0·540
10 to 11 "	75 "	3·472	0·795
11 to 12	54 "	2·410	0·631
12 to 1 p.m.	87 "	3·625	1·061

December 24th.			
Hour.	Quantity.	Sugar.	Urea.
1 to 9 a.m.	3950 c.c.	154·296	31·235
9 to 10 ,, ...	110 ,,	4·741	1·001
10 to 11 ,, ...	135 ,,	5·443	1·593
11 to 12	90 ,,	3·629	1·116
12 to 1 p.m.	78 ,,	2·635	0·982

Another intermission then occurred in the urine, which was again collected from 4 p.m. to 8 p.m., December 26th. Then again it was collected from 11 a.m. to 3·30 p.m., December 27th. On this last day the amount of urine was very small, and then the chloride of sodium was allowed for. The amount of sugar also was so small that it was difficult to tell when all trace of blue colour was gone, and some mostly remained.

December 26th.			
Hour.	Total Quantity.	Total Sugar.	Total Urea.
4 to 5 p.m.	120 c.c.	5·769	1·224
5 to 6 ,, ...	128 ,,	6·666	1·190
6 to 7 ,, ...	205 ,,	9·318	1·640
7 to 8 ,, ...	154 ,,	6·875	1·185
December 27th.			
11 a.m. to 1·30 p.m. ...	133 c.c.	3·228	1·436
1·30 to 2·30 p.m..........	45 ,,	0·852	0·567
2·30 to 3·30 ,, ...	50 ,,	0·961	0·700

5. *Temperature.*

Charts are given to show the variation in the temperature each quarter of an hour, judged of by a thermometer placed in the axilla. In the columns above, the amount of sugar, urea, chloride of sodium, and water, is given hourly, and written down opposite to the time at which the urine was collected. The hourly amount is always given.

Hudson.

October 24th.—The patient finished dinner at 1·10. He ate heartily of fish, bread, and potatoes, and took half a pint of porter. He took nothing more till tea, which he completed by 6·45, and ate heartily of meat and bread and butter; after this he took nothing till after 10 p.m.

26th.—Breakfasted exactly at 9; ate very heartily. At 4 a.m., previous, he had taken some tea and a little bread and butter.

27th.—He commenced his breakfast at 7; it consisted of tea and toast, the tea with milk and sugar; breakfast very good.

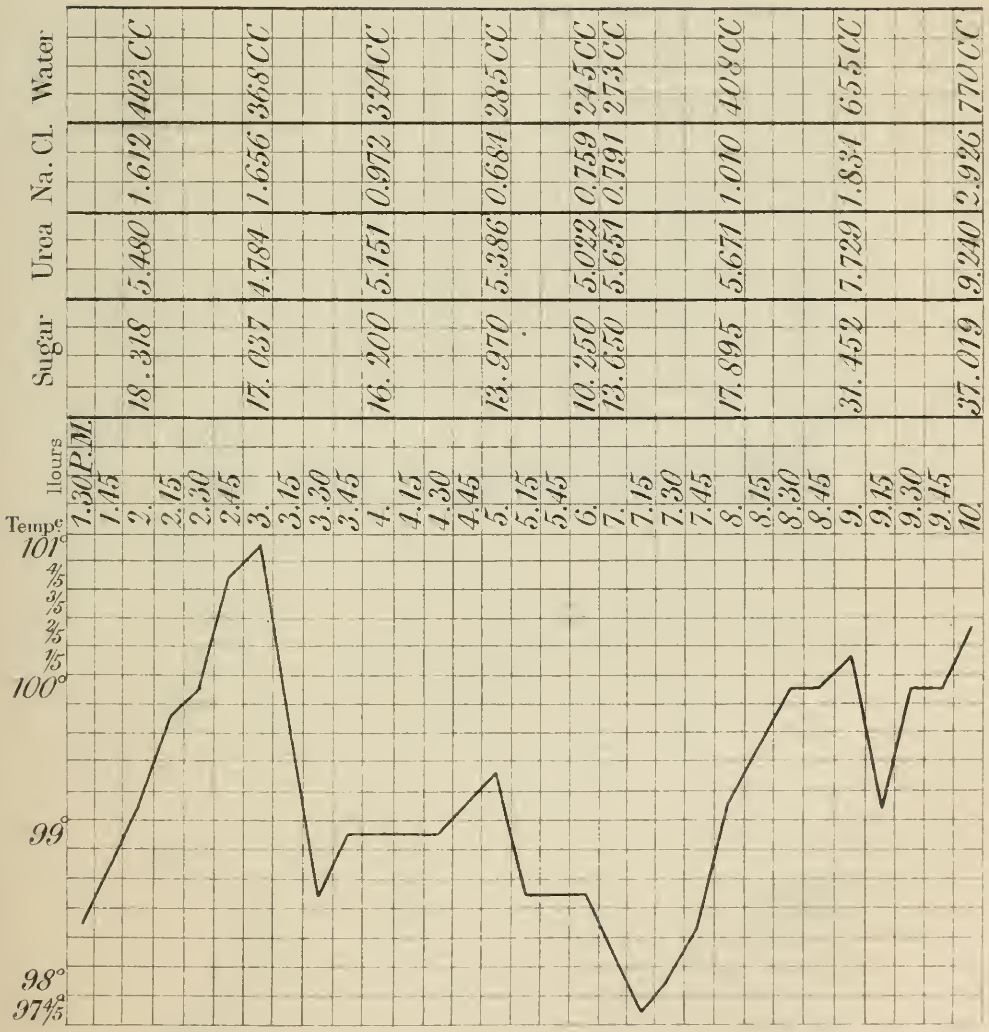
28th.—Breakfasted at 7·30; he ate six eggs for breakfast, and less bread than usual. Dined, at 1, on meat, bread, and potatoes, and half a pint of beer; he ate heartily. Tea at 5, very good.

At 6 p.m. the temperature rose suddenly to $100\frac{4}{5}^{\circ}$, and, in a few minutes, fell again to $99\frac{1}{5}^{\circ}$. The rise followed after a cup of tea, by no means hot. A similar rapid rise, and subsequent fall, after drinking, was noticed on another occasion.

These charts require no remarks, as they speak for themselves. Suffice it to say, that the temperature of the axilla was taken by an accurate thermometer, graduated into fifths.

TEMPERATURE IN DIABETES MELLITUS.

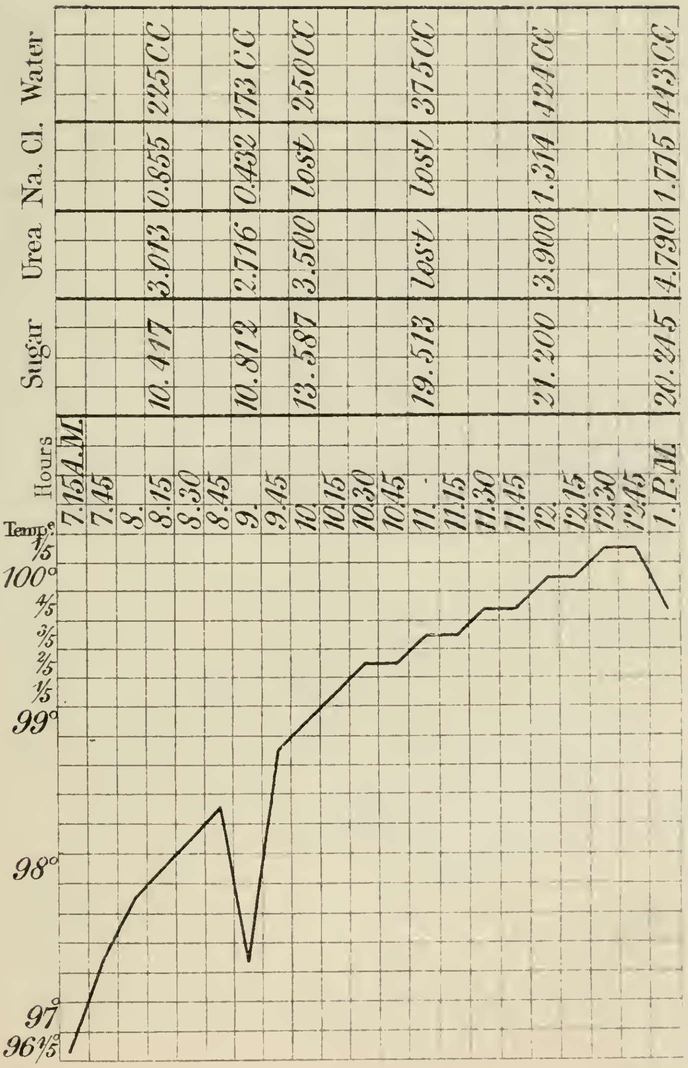
OCTOBER 24TH.



Hours	Sugar	Urea	Na. Cl.	Water
2.	18.318	5.480	1.612	403CC
3.	17.037	4.784	1.656	368CC
4.	16.200	5.151	0.972	324CC
5.	13.970	5.386	0.684	285CC
6.	10.250	5.022	0.759	245CC
7.	13.650	5.651	0.791	273CC
8.	17.895	5.671	1.010	408CC
9.	31.452	7.729	1.831	655CC
10.	37.019	9.240	2.926	770CC

W West lith.

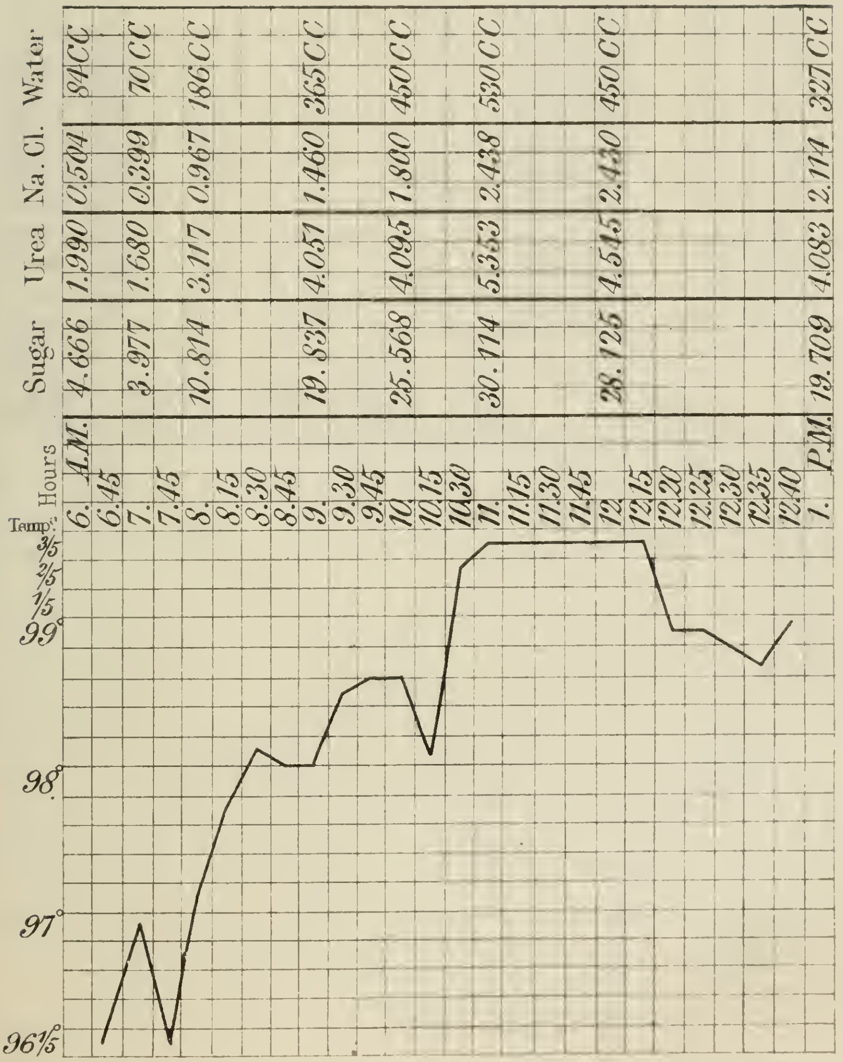
OCTOBER 26TH



Hours	Sugar	Urea	Na. Cl.	Water
7.15 A.M.				
7.45				
8.				
8.15	10.417	3.013	0.855	225 CC
8.30				
8.45				
9.	10.812	2.716	0.432	173 CC
9.45				
10.	13.587	3.500	lost	250 CC
10.15				
10.30				
10.45				
11.	19.513	lost	lost	375 CC
11.15				
11.30				
11.45				
12.	21.200	3.900	1.314	124 CC
12.15				
12.30				
12.45				
1. P.M.	20.245	4.790	1.775	443 CC

W. West, lith.

OCTOBER 27TH



Hours	Sugar	Urea	Na. Cl.	Water
6. A.M.	4.666	1.990	0.504	84 CC
6.45				
7.	3.977	1.680	0.399	70 CC
7.45				
8.	10.814	3.117	0.967	186 CC
8.15				
8.30				
8.45				
9.	19.837	4.051	1.460	365 CC
9.30				
9.45				
10.	25.568	4.095	1.800	450 CC
10.15				
10.30				
11.	30.114	5.353	2.438	530 CC
11.15				
11.30				
11.45				
12.	28.125	4.545	2.430	450 CC
12.15				
12.20				
12.25				
12.30				
12.35				
12.40				
1. P.M.	19.709	4.083	2.114	327 CC

W. West, lith.

TABULAR STATEMENT OF SEVENTY-TWO CASES

OF

HÆMATEMESIS;

WITH REMARKS.

BY

C. HANDFIELD JONES, M.B., F.R.S.

COMMUNICATED BY

H. BENCE JONES, M.D., F.R.S.

Received June 5th.—Read June 26th, 1860.

An examination of the circumstances under which any striking symptom occurs is of advantage, as leading the mind to estimate more truly the value of the symptom, to understand more fully its possible import, and to enable us thus, from what is seen and evident, to infer more correctly what is obscure and conjectural. The phenomenon of gastric hæmorrhage is one to which these observations are very applicable; it is never an unimportant occurrence, often one of fearful omen, and it is, in all cases, of great moment to be able to assign a sufficient and probable cause for its appearance. While I always feel reluctant to lay much stress upon statistics, especially in the matter of treatment, I think it can only be of advantage to record, numerically, the results presented to us by a simple observation of actual facts.

The accompanying tables have been constructed from the records of 2500 cases, observed by me, during about the last seven years, in public and private practice. These have been selected from the whole number I have had to do with, on the ground of their possessing some point of interest worth recording. Though selected cases, they are, by no means, to be considered as forming a special group, but rather as a fair representation of the ordinary occurrence of disease. I believe I shall not err widely if I assume that the number of selected cases is about one fourth of the whole mass, and that, among the unrecorded, the symptom I intend to examine has rarely, if ever, occurred; as, if it had been otherwise, it would probably have arrested my attention. On a rough calculation, therefore, it may be stated that, among 2500 selected cases of all kinds, and 10,000 unselected, the sign of hæmatemesis was met with in about 72.

Of these 72 cases, 53 were females, 19 males—a proportion of nearly 3 to 1.

The hæmorrhage occurred—before the age of 20 in 2 males and 3 females; from the age of 20 to 40 in 9 males and 36 females; after the age of 40 in 8 males and 14 females.

In 28 cases indigestion is mentioned as occurring a month, or longer, prior to the hæmatemesis. In 12 cases it is stated that indigestion was absent, or very slight.

In 40 cases the existence of gastric ulceration seems to be more or less certain.

In 32 cases its existence is more or less doubtful.

Of the former class there are 17 in which great improvement was effected by the steady and decided use of tonics: viz., Cases 3, 5, 8, 9, 10, 11, 12, 14, 16, 17, 19, 22, 23, 24, 33, 50 and 57.

In 3 of the latter class (Cases 20, 62, and 69) the vomiting of blood occurred during epileptoid fits, and gastric disorder was absent in 2 of these. In 3 others (Cases 31, 36, and 65) the liver was in a state of cirrhosis, as shown by the autopsies. In Case 35 the liver was in a state of acute yellow atrophy. In Case 44 the spleen was greatly enlarged, and there was ascites. In 2 cases (49 and 72) the presence of cancer in the stomach was highly probable. In 7 cases (16, 18, 21, 29, 30, 47, and 66) there were peculiar nervous phenomena, not in any way resulting from the hæmorrhage, but rather appearing to stand in the position of a promoting cause. In Cases 1 and 42 acute rheumatism appeared to have some relation to the hæmatemesis. In Case 59 rheumatism was associated with purpura. In 5 cases (24, 27, 45, 52, and 60) hæmatemesis had a distinct relation to the catamenial periods.

According to the above general estimate the frequency of the occurrence of hæmatemesis may be stated as 0·72 per cent. of all cases taken indifferently, and that of gastric ulcer as 0·4 per cent. In my inspections of 100 stomachs, taken indifferently from patients dying of all sorts of diseases, I found, in 6, existing ulceration, or the remains of by-past, a fifteen-fold greater proportion than is expressed by the above figures. Of course it would be absurd to expect the proportions in the two series to be similar, the one taking account only of the gravest issue of the gravest diseases, the other dealing chiefly with the slighter and more ordinary affections. For this very reason I think the latter may be of some value.

The chief practical points, which examination of the above cases brings to view, are—

1st. The number of cases met with, in which the existence of gastric ulceration is a matter of great uncertainty, in which one cannot avoid asking one's self whether the hæmorrhage may not be simply the analogue of common epistaxis. That such is possible, even where the bleeding is copious, is proved by the record of a case given by Dr. Brittan. A female, æt. 29, after persistent stomach symptoms, resembling ulceration, attended twice with considerable vomiting of blood, the catamenia being generally regular, dies at last with severe head pain. At the autopsy the viscera were all found healthy excepting a cyst in the fourth ventricle of the brain. The conclusion drawn by Dr. Brittan is doubtless correct, that irritation of the roots of the pneumogastric nerve by the cyst was the cause of the gastric symptoms. He has met with two other similar cases. In those above mentioned, where nervous debility and disorder were the prominent symptoms, and seemed to be rather the cause than the consequence of the hæmorrhage, it seems to me highly probable that the hæmatemesis resulted from a temporary congestion of the mucous membrane, induced by paralysis of the vaso-motor nerves of its arteries. Cases 18, 29, 30, 66, were very marked instances of this kind. Gastric hæmorrhage has occurred in persons who have ascended to a great altitude, where the atmospheric pressure was much diminished, and of course the strain on the internal surface of the vessel *pro tanto* increased. This is an analogous condition to that of vaso-motor nerve paralysis.

2d. The number of cases, in which all complaint of dyspepsia was either absent or so slight, that it would have been impossible to distinguish it from that attendant on slight gastric catarrh or neuralgia. In the great majority of cases, it certainly does appear that there is very little that is at all distinctive in the subjective symptoms attending on gastric ulceration. In fact, one can hardly avoid questioning, whether they are not rather the result of neuralgia, or of the concomitant catarrhal inflammation, which is so often present that Rokitansky affirms it is constant. This view is still further confirmed by the occasional occurrence of cases in which no symptom of indigestion whatever, or gastric pain precedes the sudden outburst of hæmorrhage, or fatal peritonitis, announcing perforation of an artery, or of the wall of the stomach. In some cases, certainly, the symptoms are strongly marked, and the nature of the malady very apparent. Persistent, wearing, gastric or dorsal pain, increased by food, emaciation, pallor, a sunken morose aspect, with repeated attacks of hæmatemesis, leave no doubt as to the nature of the disease; but, between these typical cases, and those in which there is an utter absence of all gastric uneasiness till the hæmorrhage or perforation occurs, there is every shade of variety to be met with. In particular, the too common error deserves to be noticed that severe pain is a reliable sign of ulceration, and the more so in proportion to its severity. *Severe* pain is much more often a sign of mere gastralgia than of actual organic disease, especially if it be paroxysmal.

3d. The great benefit of a tonic plan of treatment steadily carried out. In several of the cases recorded the effect was very marked. Of course, it is essential that the stomach should be in a state in which the tonics are well borne; if this is not the case they must prove injurious. The coexisting catarrhal inflammation may require appropriate treatment, perhaps some leeches, or a blister, but as soon as tonics can be safely commenced, they should be employed, and then steadily continued as long as there is a manifest gain in general health and strength. We can have no positive assurance of the healing of the ulcer, we can only proceed on probabilities. Now it is notorious that an ulcer, whenever we can see it, is most favorably circumstanced for healing, when the system is in full health and vigour, and all impediments to a free circulation of blood in the part are removed. If, therefore, we have reason to believe that the portal circulation is free, and that there is no active inflammation of the gastric mucous membrane, we have no more pressing indication than to improve to the utmost the general power, in order that the nutrient and reparative processes may go on as actively as possible. We shall see this still more clearly, if we hold in mind that the ulcerative process in its simple and uncomplicated form is essentially dependent on local decay, not in any wise on increased action. The perforating tendency especially marks this asthenic quality. It is the clean cut ulcers, with level edges showing no trace of exudation, that most often produce perforation both in the gastric wall and in the cornea.

One very essential part of the tonic treatment will consist in the administration of nourishing food. This, of course, requires judgment; if the nutriment is not easily digested, if it causes pain and uneasiness, it will aggravate the local disease; but if it is borne well, it will act beneficially by supplying abundantly the materials of healthy blood, and so promoting nutrition. To ensure easy digestion an hour at least should be devoted to complete repose after each meal, and the process may be further assisted if necessary by pepsine, or lactic acid. All immoderate distension of the stomach should be avoided whether by copious or bulky food, or flatulent secretion.

Tabular statement of seventy-two cases of Hæmatemesis.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
1	S. H.	F.	22	22	Rheumatic fever; amenorrhœa; health generally good	On recovering from rheumatic fever had hæmatemesis recurring every day for a month, ceased the last week; pain afterwards in right hypochondrium and between shoulders, none after food	Improved much under tonics, could eat her food comfortably after seventeen days of treatment, not seen since.
2	E. B.	F.	29	28	None stated	A teacupful of blood vomited on several occasions in course of nine months; epigastrium tender, food causes pressing pain; interscap. pain; menorrhagia	Attended only one week.
3	L. B.	F.	23	23	Was rather ailing for three or four years, but digestion pretty good. Pneumonia eighteen months before hæmatemesis	Gnawing pain after taking food, which lies heavy; recurring diarrhœa; anæmia; debility; a year after was seen again and treated for weak digestion	Treated steadily for three months by tonics, sedatives, and cod-liver oil. Improved greatly, was better than she had been for four years. No recurrence of hæmatemesis one year later.
4	M. W.	F.	27	27	Widow, confined six weeks ago, extremely overworked; indigestion for one month; nausea and epigastric tenderness before hæmatemesis	Most marked debility, overwork continued; thirst, nausea, retching and anorexia; temporary benefit from tonics; recurrence of hæmatemesis	Not known.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
5	M. S.	F.	40	40	Sickness for fourteen days before hæmatemesis	Hæmatemesis to nearly Oij four months before admission, sickness three or four times a week since; epigastric tenderness; pain and vomiting two hours after food; appetite poor; amenorrhœa four months; debility; syncopic attacks	Treated by astringents and sedatives with oil, after by nitric acid and oil; improved very greatly. Treated again two years and a half after for indigestion with great benefit. Hæmatemesis had not recurred.
6	M. Wh.	F.	40	40	Very subject to rheumatism; no definite mention of prior dyspepsia; amenorrhœa succeeding menorrhagia	Ill five months; hæmatemesis to Oj last five days; much epigastric and lumbar pain, food either vomited or passed per anum quickly; continued thirst; attacks of syncope	No steady treatment.
7	C. F.	F.	36	36	Bad digestion two years; much trouble; a brother died at same age with hæmatemesis	Ill six weeks, attacked while at work with violent pain in chest, vomiting of blood, and syncope; hæmatemesis has recurred twice since; is trembling, weak, and giddy; meat causes pain; lungs emphysematous	Some recurrence of hæmatemesis after fourteen days; improved to some extent under tonics. Treatment broken off by non-attendance.
8	Ch. Ald.	F.	24	24	None till eight days before, when had nausea and pain in stomach, being affected by bad smell from a sick-room; overworked; catamenia scanty	Vomited about Ovij of blood in twenty-four hours; pain in stomach ceased after hæmatemesis set in; digestion feeble, but no remarkable symptoms except occasional nocturnal attacks of pain and diarrhœa	Treated by astringents, tonics, and sedatives, with much advantage. Had much improved when she left for country after five weeks.

9	S. B.	F.	24	24	Overwork; pain after solid food, and vomiting of same; not strong for four years; catamenia ceased after one day, two to three days before hæmatemesis	After sick head-ache for two or three days took two pills, and immediately vomited, first, mucous matter, &c., and then Oss of pure blood. No emaciation	Under treatment by nitric acid and gentian about two months. Improved greatly.
10	M. Pr.	F.	34	32	Stomach disorder on and off for fifteen years	Pain at chest; vomiting of all food directly, or in an hour and a half, in paroxysms lasting two to four weeks; in intervals can retain her food fairly; no marked epigastric tenderness; hæmatemesis recurred five weeks after admission	Under treatment four months and a half, improved a good deal under tonics and sedatives.
11	O. M'C.	M.	59	57	Same as after hæmatemesis	Heartburn, very severe pain, worse after eating; no epigastric tenderness; lungs emphysematous; apyrexia	Treated seven weeks, much improved by tonics.
12	S. D.	F.	30	28	Got out of health by coming to reside in London; had indigestion and a sense of distension at stomach for two months, lost strength and flesh; after great pain at left side for two or three days faintness occurred, and then hæmatemesis to a washhand basin full; the bleeding continuing fourteen days	Often has pain at lower abdomen, sensation of gathering at lower sternal region, then violent retching and rejection of clear water; food does not cause pain, but turns acid and is vomited often; interscapular pain; no epigastric tenderness; catamenia regular; ceased for some time after hæmatemesis	Apparent complete recovery by tonics and change of air. Recurrence of indigestion four months after, but no hæmatemesis.
13	F. Pr.	M.	62	44	Not mentioned; second attack of hæmatemesis eighteen years after first	Health pretty good, frequent "bilious attacks." Frequently has pain after eating, food turning acid and being often vomited; immediate relief from soda; sharp epigastric pain, but no tenderness; has also had pain in loins, and sense of heavy weight at back of neck	Improvement commenced under tonics. Ceased attendance.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
14	F. Pab.	F.	33	53	Always ailing, subject to catarrh	Epigastrium tender (hysteric?) solid food does not digest well, but pain is not more after eating	Treated three months steadily with astringents, cod-liver oil, acids, and quinine. Digestion became quite comfortable and strength improved.
15	S. P.	F.	27	22	No disorder of digestion before hæmatemesis, ever since it has been uncertain and failing; first amount = Ovj in fourteen days	Hæmatemesis after four years and three quarters; has now choking pain after food, followed in two hours sometimes by severe interscapular pain; crampy pain round shoulders; poverty; seven months and a half after seen again with vomica in right apex	Treated about one month; some benefit from steel and opium with blister to epigastrium. Bismuth useful.
16	A. J.	F.	34	34	Continual pain at chest; before this had inflammation of eyes. Oij of coagulated blood vomited six weeks before admission	Catamenia too profuse, regular; violent inward pain referred to lower sternal region, not increased on pressure nor by food; feels much better when quiet; after any exertion vomits up clear water and has increase of pain; benefit from stimulants; bad appetite; no thirst; severe relapse of pain and vomiting after over-exertion	Benefit from Citr. iron and quinine. After relapse, apparent cure by strychnia and Ferri and Mang. Carb.
17	F. C.	F.	40	36	No symptoms of indigestion. Recovered from hæmatemesis by going into country, and has been well ever since	Mid-sternal pain passing to between shoulders, sometimes very severe for one or two hours; always worse after food, most after liquids. Tea, and bread and butter cause as much pain	Treated by citrate of iron and quinine for about one month, when she got apparently quite well.

						as meat; brandy and water, and fresh air give relief. Pulse feeble; circulation languid; tongue natural. Lifting a weight causes much pain at epigastrium	
18	E. F.	F.	35	35	Extreme debility, fainting several times a day for eight days; then rejection of blood with nausea, but no vomiting, which has been going on a whole month	Extreme exhaustion a quarter of an hour after food, which lies heavy, but does not cause much pain. Has copious cold sweats at night; had several recurrences of fainting or leipothymia, and of rejection of blood once to Oiss; copious leucorrhœa	Much benefit from iron, quinine, and strychnine, with change of air.
19	E. Ch.	F.	27	27	Suffered with bad digestion four years. She and all her family are subject to bilious attacks, occurring every one or two months, and lasting two or three days. In these she vomits watery fluid and food, and the motions are blackish. In one of these had hæmatemesis to Oiij	General debility, and increased appetite since hæmatemesis; not much pain after food; slight epigastric tenderness	Treated at first by nitro-muriatic acid and Taraxacum, with small repeated doses of blue pill for one month, then for two months with iron and quinine; under this improved very greatly; was in better health than for four years.
20	C. P.	M.	21	21	Said to have had enteritis and peritonitis eighteen months ago; continued afterwards well for one year; had then an epileptic fit and vomited Oij of blood; had two fits before; has suffered from pains in bowels since	Aching pain relieved by leeching epigastrium, increased by food, flatulence. Food rejected by a kind of rumination; no discoverable disease of thoracic or abdominal viscera; severe frontal headache when bowels are confined	No notable improvement.
21	S. Ar.	F.	45	35	Catamenia recur every fourteen days and last seven. Her family are prone to hæmorrhage. If she gets the least cough expectorates blood	Suffers with recurring eczema (slight), neurolysis, bleeding hæmorrhoids, and menorrhagia. No special stomach symptoms; not at all anæmic	Great benefit from strychnia and tannin.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
22	W. S.	M.	35	35	One year ago temporary paralysis from a fit; cough last winter; no indigestion before hæmatemesis	Strained himself while lifting a weight; copious hæmatemesis followed immediately; has had indigestion since. Blood passed in stools; lungs sound	Great improvement under bismuth and tannin, strychnia, iron, quinine, and Ol. Morrh., gaining strength every day at seven weeks date from commencement of treatment.
23	E. M.	F.	32	32	Always healthy. Last four or five months has suffered much from weak digestion and palpitation of heart; of large, lax make; catamenia regular	Hæmatemesis to Oij two days before admission; has nausea; much thirst; no appetite; cool skin; feeble pulse; tongue nearly natural; heart and lungs sound; epigastrium soft and painless	Bismuth and tannin of much benefit; took quinine afterwards. Left hospital to go to country in three weeks; was very weak, but took fish, an egg, &c., comfortably.
24	M. A. W.	F.	25	25	Food has not digested well for three or four months before hæmatemesis; felt faint and sickish seven days before	Hæmatemesis occurred at a catamenial period; the flow stopped after two days in afternoon, and the blood (Oiss) came up at night. Some indigestion, but no epigastric tenderness, or interscapular pain; heart and lungs normal	Treated nearly three weeks with quinine, strychnia, and iron and quinine; gained much strength; food digested quite well. Catamenia absent last period.
25	J. B.	M.	50	43	Never had any stomach ailment at all before first attack of hæmatemesis; of short, stout make	Repeated attacks of hæmatemesis during seven years; once vomited five measured quarts. Suffered with severe dyspepsia, flatulence, loss of appetite, retching, purulent urine, anæmia, debility; vomit has several times contained sarcinæ	Benefit from Argenti Nitrat. and lactic acid. No steady treatment yet.

26	A. S.	F.	43	43	Ill nearly a year, after some heavy laundress-work felt she had strained her left side, and ever since has had pain there, and no appetite. Has now exceeding tenderness all about the epigastrium, and both hypochondria, as well as both sides of chest at lower part. The pain destroys all rest, is relieved somewhat by hot brandy and water. She cannot touch solid food, and is extremely emaciated, with arid, raw, aphthous tongue, and a feeble small pulse. No fever or thirst. Distinct pulsation in front of upper abdominal aorta; three months ago there was a marked bruit all along abdominal aorta, and audible even in sacral region. Hæmatemesis occurred about fourteen days before death, together with bloody stools, and lasted seven days. Died exhausted		No relief from large doses of opium; posterior wall of stomach most extensively destroyed by ulceration; a large splenic vein and two large splenic arteries lay, open-mouthed, in the ulcer; stomach adherent to left lobe of liver and pancreas.
27	Ph. C.	F.	35	35	No disorder of digestion; hæmatemesis during a catamenial period, toward its close; catamenia irregular, sometimes excessive, and then absent for long intervals	Suffering with neurolytic disorder; varicose veins in leg	Some benefit from bismuth; tonics failed.
28	A. D.	F.	20	20	Ill nine months, suffering with pain in epigastrium and lower sternal region; made much worse by food, especially meat; tenderness on ressure; nausea; no appetite. Has vomited blood in small quantities two or three times every week during all her illness; it has been mixed with phlegm. Food vomited before admitted. Has occasional chills, and heat flushes, and nocturnal sweats. Not emaciated notably		Had ice, and afterwards pepsine; soon recovered; gained four pounds in one week.
29	M. A. L.	F.	42	42	No illness before her present. Suffering for thirteen weeks with pain of right knee, no swelling. Blood has been brought up last ten days, it is not vomited, but rises up into mouth. Some sickness in morning. Pain at epigastrium. Appetite bad. The rejection of blood continued to recur frequently for four or five months. The knee pain was so severe as to suggest amputation		Rejection of blood ceased under the use of carbonate of iron; knee pain gradually subsided.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
30	J. O'C.	F.	35	35	Hæmoptysis and bloody stools at æt. 14; catamenia regular; excessive leucorrhœa, three months until last two; neuralgic pain of right foot and shoulder; febricitation; sensation of nausea	Impaired digestion; rejection of blood without vomiting in afternoon and night of most days to ℥iv or ℥v daily; lungs and heart sound; aguish symptoms; occasional vomiting of blood in larger quantity; loss of cerebral power (temporary), and stupor	Much benefit from quinine for a time; subsequent failure of all remedies. Complete recovery for more than two years, after a voyage to Ireland, and short stay there. Subsequent recurrence of hæmatemesis.
31	H. R.	M.	49	49	None specified. Hæmatemesis occurred after a fall on epigastrium; quantity = Oj	Ascites, anasarca, sign of valvular disease, dyspnœa, breathlessness, cough. Death	Heart enlarged, aortic and mitral valves diseased; lungs engorged; liquid in both pleuræ; liver cirrhosed; kidneys somewhat atrophied.
32	E. W.	F.	55	55	Sense of burning heat at epigastrium brought on by the least amount of food; relieved in about one hour by vomiting of watery and tenacious mucus and ingesta	℥ij of blood vomited; tenderness above umbilicus; urine deposits uric acid. Catamenia ceased	Much benefit from hydrocyanic acid, Arg. Nitr., bismuth, and afterwards tonics and astringents.
33	E. Pr.	F.	27	27	Dyspepsia, severe pain after food, relieved by hydrocyanic acid and careful diet	Intense pain after food; extreme epigastric tenderness; comparative ease when stomach was empty. Vomiting and some hæmatemesis occurred	Cured by tannic and nitric acid, sedatives and counter-irritation having failed.
34	J. H.	M.	58	55	Suffered more than twenty years with occasional attacks of pyrosis and epigastric pain coming	Same as before hæmatemesis. Great benefit from two voyages to Madeira; subsequent recurrence of bad symp-	Post-m. showed perforation of stomach by an ulcer and effusion with peritonitis.

					on one hour after meals, sometimes attended with vomiting. Symptoms increased by anxiety	toms. Death in forty-eight hours after sudden occurrence of perforation	
35	M. A. L.	F.	25	25	Continual vomiting of yellow matter; deep jaundice. Delirium, stupor, passing into coma, in which she died. Copious hæmatemesis shortly before death		Liver in state of acute yellow atrophy.
36	C. Q.	F.	33	33	Always subject to vomiting and sick headache recurring once a month or oftener. Digestion bad for a long time; catamenia regular	Much blood vomited and passed in stools; hepatic resonance diminished; ascites; diarrhœa; urine albuminous. Died	Stomach healthy, liver cirrhosed. Spleen large. Copious ascites.
37	J. D.	F.	40	39	Indigestion for years; hæmatemesis to Oij one year before	Appetite lost; pain from food passing from left side to epigastrium; tongue coated; epigastrium tender	Much benefit from calomel, with aperients, and nitro-muriatic acid with taraxacum.
38	M. A. K.	F.	40	40	Severe pain, and heat of epigastrium, rising in throat, chiefly after food. Vomits thick phlegm and froth. Hæmatemesis slight		Much benefit from sedatives, astringents, and tonics.
39	A. W.	F.	21	17	Hæmatemesis to Oj	Left side pain increased by exertion; slight rejection of blood in mornings; digestion bad; catamenia regular; rejects more blood during periods of catamenia	Slight benefit from tonics and antispasmodics.
40	Mr. Hol.	F.	40	34	None stated	Was ill a good while after copious hæmatemesis; digestion not strong since, but otherwise well.	——
41	M. Fitz G.	F.	60	60	Mental trouble and poverty; stomach irritable	Pyrosis; pain at stomach; indigestion; syncope	Improved under use of bismuth, alkalies, and muriatic acid.
42	El. B.	F.	14	14	Articular rheumatism, and pericarditis	Epistaxis; slight hæmatemesis; delusions	Ordinary treatment of rheumatism. Recovered.
43	M. C.	F.	36	36	Cholera soon after confinement; ailing ever since; disorder of stomach (slight) before hæmatemesis; bad appetite	Serous effusion in left pleura removed by paracentesis	Recovered.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
44	W. St.	M.	26	26	No ague. Digestion generally good, but has been impaired till lately	Considerable enlargement of spleen; copious hæmatemesis three times in about four months; liver not enlarged; anæmia; ascites	Remained in about same state.
45	M. Ed	F.	30	—	Is often sick, and vomits very black stuff at the catamenial period, and then only. Ill two years with pain in right side, and very bad digestion		Relief from sedatives.
46	M. A. T.	F.	60	—	Ill two or three years, suffering at intervals with severe paroxysm of gastric neuralgia; after emetics has vomited Oss of blood mixed with phlegm		Great benefit from quinine and iron and potass. iodid.
47	Mrs. D.	F.	50	41	Disorder of stomach, pain after eating. Vomited two quarts of black blood	Excessively hyperæsthetic, suffering with all sorts of nervous disorders	Some benefit from bismuth.
48	S. L.	F.	4½	4½	Ever since severe attacks of pertussis, æt. 1½ year, has been subject to vomiting of food, latterly a little blood has come up with it	Health good. Has pain about umbilicus. The vomiting chiefly occurs at night; blood in small quantities frequently vomited	Some benefit from acids and sedatives.
49	H. T.	M.	44	41	Debility, vomiting of food, which lies heavy. He drank a good deal	Same symptoms. Husky cough; gradually sank and died. I regarded his disease as cancer of the stomach	No medicine of any avail.
50	C. C.	M.	29	29	Ill ten weeks with nausea; much flatulence, and vomiting of green bile, occasionally mingled with blood in small quantity. The vomiting occurs after eating most days. Is generally healthy		Strychnia of much benefit.
51	C. K.	M.	34	24	During first two years of illness vomited small quantities of blood twice or thrice. Of late years he has vomited larger quantities, and oftener. Last two years has vomiting every day, quantity varying from ℥ij to ℥x, usually vomits about 4 a.m., sometimes brings up acid water and phlegm before the blood. Has constantly a burning hot sensation at		Bismuth, quinine, hyposulphite of soda, and leeches to the epigastrium failed to be of any service.

					the epigastrium, much aggravated before and after the ejection. Much epigastric tenderness. Had ague thirteen years ago. Tubular and weak breathing at upper parts of both lungs. No sarcinæ in vomit. No tumours to be detected. Not much blood was vomited while in hospital		
52	C. T.	F.	23	23	Has catamenia every three weeks, and too profusely. At these periods blood comes up from mouth by retching, with nausea and faintness. Has bad appetite, and pain after eating		Much benefit from strychnia and quinine and iron.
53	J. Hd.	F.	60	60	From infancy to adult age subject to severe epistaxis. Poorly and weak for some days. Deadly faintness occurred twice in one morning, and in about two hours complete and prolonged syncope. Hardly knows what indigestion is. Lives in an aguish district	After a dose of castor-oil copious and repeated hæmatemesis; blood passed also per anum	Bleeding stopped by styptic powders with opium, a course of gray powder after. Health quite restored by nursing, no relapse to this date.
54	Cath. Hd.	F.	67	61	Stomach delicate; vegetables disagree. Pallor and debility before last attack, which was at first moderate, but caused much prostration, and was succeeded in twelve or thirteen weeks by a most copious hæmatemesis, which all but destroyed life	Has remained ever since in a very feeble state	No return of hæmorrhage for four years; appetite good.
55	M. Wt.	F.	30	30	For three months has been bringing up blood with straining effort, about half a cupful a day for last fourteen days. No pain after food, but violent pains round waist every now and then; great poverty and debility. Has cold sweats and shivers. Chest healthy. If she cannot get food at 11 a.m. syncope occurs		Benefit from being better fed.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
56	R. Ch.	F.	45	38	Ill eight months, suffering with pain at epigastrium, between shoulders, and almost everywhere constantly, but it is worst after food. Has had copious hæmatemesis twice. Has had eight children. Suckling until last hæmatemesis. Great sense of sinking at epigastrium. Urine as clear as water		Temporary benefit from bismuth and Fer. and Quin. Citr.
57	A. Dav.	F.	67	67	Chronic bronchial catarrh; emaciation; debility	Was attacked with sickness and giddiness in the night, and vomited blood; had much pain in chest and abdomen	Much benefit from gallic acid and tonics.
58	S. F.	F.	65	65	Copious hæmatemesis some months ago	Symptoms of gastric and bronchial catarrh	Much benefit from tonics and cod-liver oil.
59	Mr. —	M.	40?	40 ?	Rheumatism of hand, knees, and ankles	Four or five days after rheumatism set in began to vomit blood and to pass some per anum. Exceeding prostration; stomach very irritable; numerous purpuric spots on thighs	Benefited greatly from quinine, morphia, and creasote, with tannin; but relapsed in a while, and died in a state of "typhus fever."
60	E. B.	F.	29	29	On three occasions has had vomiting of blood, not very copious, during the presence of the catamenia. Has symptoms of uterine congestion and relaxation. Has aborted three times. No complaint of indigestion		Benefit from strychnia and iron.
61	M. D.	M.	53	53	Getting ill last six months gradually; vomited a large quantity of blood	Stomach very irritable; emaciation extreme; lungs emphysematous; abdomen greatly shrunk	No amendment; went out in same state.
62	Eug. M.	M.	28	28	Has at various intervals paroxysms of opisthotonos, in some of which he vomited a small quantity of blood. Was first attacked two years ago, at Calcutta		Had large doses of quinine without benefit.
63	W. L. H.	M.	40	40	Urine very dark coloured, containing blood (?); vomited some clotted blood a few days ago; symptoms are those of severe lumbar rheumatism chiefly. Liver congested (?)		Very much benefit from Ammon. Murias and Pot. Iodid.

64	J. H.	F.	24	24	Ill three days, at first was sick and vomited much blood, passed blood per anum five weeks ago, has no piles. Suffers from very severe cramp-like pains in abdomen, thirst, anorexia, bad cough; right hypochondrium tender		Much benefit from leeches to abdomen. Gray and Dover's powder; subsequently iron and quinine.
65	Dr. W.	M.	46	46	Considerable ascites and anasarca; urine very highly albuminous	Very frequent vomiting; blood vomited and passed per anum. Gradual asthenia and death	No treatment of any avail. Liver in an extreme degree of cirrhosis.
66	Ch. C.	M.	10	10	Symptoms of nervous prostration and loss of speech were preceded by retching, in which he brought up nearly Oss of blood. Epigastrium tender		Recovered with iron and quinine and country air.
67	M. Fr.	F.	49	49	For nine months before hæmatemesis felt pain at epigastrium and left side, and between shoulders, increased after food, and has been drooping	Great pain at epigastrium. Has passed per anum a very large quantity of black blood and vomited much red. Tongue coated; skin cool; pulse weak	Improvement from acids and astringents; treatment not continued.
68	J. P.	M.	31	21	Hyperæsthetic, very anæmic, very weak, severe præcordial pain, food lies heavy, but has no particular pain after eating. No epigastric tenderness. Has had hæmatemesis four times in ten years, the last time two years ago		Attended only one week; took strychnia and iron and quinine with benefit.
69	A. D.	M.	19	19	Epileptic fits and masturbation	Brought up half a tea-cupful of blood in a fit.	—
70	R. P.	F.	57	57	Not much dyspepsia before the attack	Two months ago vomited nearly Oiv of congealed blood. Has pain under left breast and in back and limbs, increased by inspiration; not much indigestion	Attended only a short time; was benefited by Fer. c. Quin. Citr. Pyrosis occurred subsequently, but no hæmatemesis.
71	E. B.	F.	44	42	Palpitation only; no indigestion. Palpitation attended with sense of strangulation, numbness of lips and hands. Eight children. Eyes have been badly inflamed; left blind	Food seems to do no good; soon feels empty and sinking. Left infra-mammary pain; catamenia too copious. No pain from food	Treatment interrupted.

Case.	Name.	Sex.	Age.	Age when Hæmatemesis first occurred.	Previous occurrences to Hæmatemesis.	Symptoms, occurrences while under observation, and some time before.	Result—Treatment.
72	M. St.	F.	69	53	Abdominal pain above and below navel, of gnawing, sickening character. Meat always vomited for a year. Stomach free from pain when empty	Same symptoms, with increasing dysphagia the last seven weeks. Can only take fluid. Frequent vomiting; great debility; emaciated.	—

ACCOUNT

OF A

FRESH DISSECTION OF A PREPARATION

OF

TUBAL GESTATION,

DESCRIBED BY DR. JOHN CLARKE IN THE 'TRANSACTIONS OF A SOCIETY FOR THE IMPROVEMENT OF MEDICAL AND CHIRURGICAL KNOWLEDGE FOR 1793.'

BY

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Received June 2d.—Read June 26th, 1860.

The following communication is intended merely as a contribution to the anatomy of cases of tubal gestation. I shall therefore refrain, as far as possible, from any theoretical opinion as to the function of the parts described, and confine myself to a simple explanation of the preparation and the drawing by which it is accompanied.

When the communication by Dr. R. Lee on this subject, which is published in the 'Medico-Chirurgical Transactions,' Vol. XLI, p. 137, was read before the Society, reference was made in the discussion which followed to the preparation on which the present paper is founded, as completely refuting the view which Dr. Lee advocated. The preparation had not at that time been completely dissected; and the further dissection which I have made of it demonstrates a state of things certainly different from the original description. As the preparation is in some respects a classical one, and is frequently appealed to by writers on this subject, it appears simply just to give publicity to the following account.

This preparation was made by the late Dr. John Clarke, and described by him in the 'Transactions of a Society for the Improvement of Medical and Chirurgical Knowledge,' vol. i, p. 215, 1793. That paper is, no doubt, familiar to most persons who have paid attention to the subject. It is illustrated by two plates, one representing the whole preparation, the other the Fallopian tube and the embryo contained in it. I need not detain the reader with any lengthy reference to that paper. It recounts the circumstances of the case—the patient dying at an early period (probably before the end of the second month) of pregnancy from hæmorrhage into the peritoneum, the effusion amounting, it is said, to a gallon; and the post-mortem examination, in which a rent was found in the Fallopian tube, filled with the shaggy vessels of the chorion, from which the bleeding had proceeded. The cervix uteri was found plugged with gelatinous matter, the body and fundus occupied by what is described as "membrana decidua" (without being further particularised); and then Dr. Clarke goes on to say, "One thing particularly deserving of notice in the present case is the formation of the membrana decidua in the uterus before the ovarian ovum had reached it. By ovarian ovum, I mean the ovum as it comes from the ovarium, or at least as it exists in the Fallopian tube, consisting of two membranes, the chorion and the amnios, which contains the liquor amnii and the embryo, but without the decidua, which membrane is formed in the uterus." The accompanying plates do not show the membranes around the ovum with sufficient distinctness to support any conclusion; but it is clear from Dr. Clarke's description, and from the state of the preparation when it came into my hands, that he believed the chorion to be covered externally by the walls of the tube.

The preparation was given by Mr. Stone, together with the rest of his splendid obstetrical collection, to the museum of St. George's Hospital. In the course of its removal thither, the embryo happened to become detached from the funis; but the latter still remains, and is clearly visible in the cavity of the amnion. On looking at the ovum, I noticed a separation between it and the wall of the tube in one part; and, by dissecting with a blunt instrument under water, this separation was easily extended around the whole circumference of the ovum (in the situation pointed out in the drawing by small pieces of bristle), except at the part where the rupture had occurred, and where the tissues were so firmly adherent that I did not attempt to separate them. This being done, the chorion was seen to be surrounded in the whole of its extent by a stout membrane, of a fibrous appearance, plainly distinguished from the chorion internally by the villous structure of the latter, as contrasted with its own fibrous texture; and distinguished from the wall of the Fallopian tube externally, inasmuch as it formed a complete circle around the ovum, and did not pass down either orifice of the tube. This is shown at D′ D″ in the drawing. On examining this membrane further, it was easy to separate it into two layers over a great part of its extent. The separation, both of the membrane from the wall of the tube, and of its two layers from each other, was effected with the greatest facility.

In microscopical structure the membrane consisted partly of ordinary fibrous tissue, partly of very firm and strong fibres, running in a straight direction, but somewhat wavy in outline, and much smaller in diameter than the fibres of unstriped muscle.

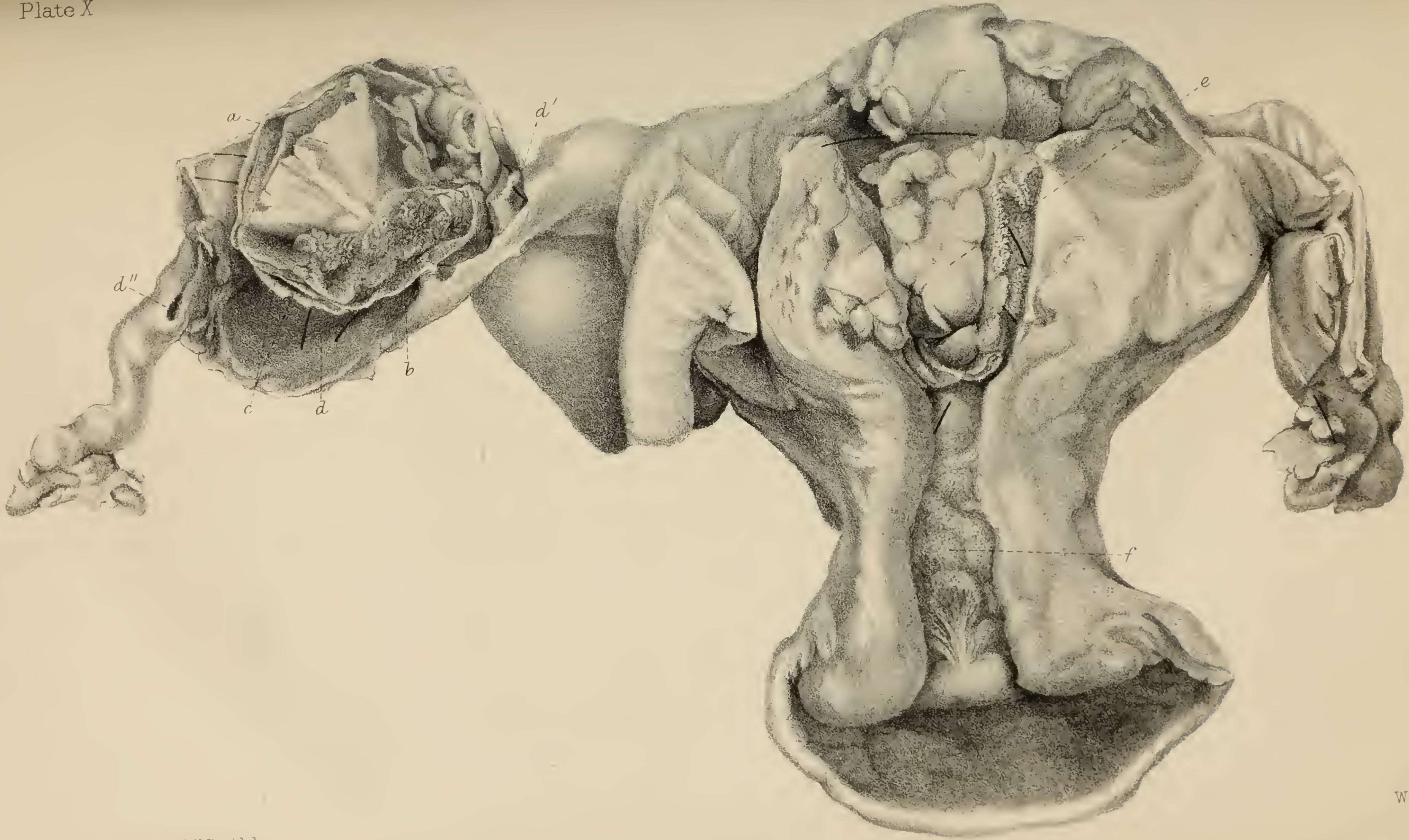
The structure which lined the body of the uterus was thick and lobular on its surface. On looking at it with a lens, small punctiform depressions could be seen in it. It was very easily raised from the surface of the uterus; as has been done on the left side of the preparation, where pieces of bristle have been placed between them, and exhibited the surface of the lining membrane roughened, but apparently unbroken. On examination of this substance with the microscope, it was seen to consist of an aggregation of cells, somewhat resembling epithelial scales, but for the most part not larger than pus-globules, and of different shapes, some being elongated, while others were round. Each had one small nucleus. They were rather granular, perhaps from partial decomposition during their long preservation in spirit. Here and there an appearance of fibrillation might be seen, but no distinct fibres.

DESCRIPTION OF PLATE.

The drawing represents the present appearance of the uterus and Fallopian tube in Dr. John Clarke's case of tubal gestation.

a. Amnion containing the funis.
b. Chorion.
c. Membrane external to the chorion, consisting of two layers.
d. Wall of Fallopian tube, showing D′ D″, its upper and lower orifices.
e. Substance lining the body of the uterus.
f. Cervix uteri, occupied by gelatinous matter.

Plate X



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CASES

OF

OBSTRUCTION OF THE VEINS OF THE LOWER EXTREMITIES,

CAUSING ŒDEMA OF THE CORRESPONDING LIMB, AND OCCURRING IN PHTHISICAL PATIENTS.

BY

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Received June 22d.—Read June 26th, 1860.

Case 1.—A gentleman, æt. 25, had symptoms of phthisis eight or ten months before his death. During the latter period of his illness he had a severe pleuritic attack; and about a fortnight before his death, œdema of the right lower extremity came on. It was attended with much pain, and the surface of the skin was shining, with considerable firmness of the cellular tissue, pitting but little under pressure.

Post-mortem examination.—About a pint of bloody serum in right thoracic cavity; a vomica in each lung, with much tubercular deposit; ulceration of the mucous coat of cæcum and colon; about two ounces of fluid in the pericardium; heart and great vessels free from disease.

The right common, external, and internal iliac veins had a cordy feel, and their cavities were filled with firm coagula slightly adherent to the vessels. This state of things was traced as far as the commencement of the femoral vein.

Case 2.—William B—, æt. 10, admitted into the Brompton Hospital, April 28th, 1852, in the last stage of phthisis. Shortly after admission he complained of pain in the left leg, which was found to be much swollen throughout its whole length, and the superficial veins on its surface much distended. The limb was not discoloured, and, although firm, it pitted under pressure.

May 21st.—Within the last few days the right leg is become œdematous, and the veins ramifying on its surface, as well as on the lower part of the abdomen, are very evident; and some of them have a cordy feel. He had much diarrhœa, gradually got weaker, and died on the 26th of May.

Post-mortem examination.—Cavities in both lungs, with much tubercular infiltration; the mucous membrane of the intestines generally much congested, but no ulcerations. The lower part of the vena cava, the common iliac, the external iliac, femoral and internal saphena veins, on both sides, were firmly distended with coagula, which were found to adhere with considerable firmness to their inner surface; the coats of the veins were somewhat thickened; the reflected and visceral portions of the pericardium were adherent.

Case 3.—M. A. S—, æt. 31, admitted into the Brompton Hospital, October, 1856. Married, and has had two children. There were marked signs of phthisis: a cavity in each lung, with much consolidation. Shortly after her admission, the left leg began to swell; she had not much pain; the skin was not red, but had a shining appearance, and pitted but slightly under pressure. It gradually extended up the thigh, involving the labia and abdominal parietes. She died November 18th.

Post-mortem examination.—Both lungs contained cavities, and were infiltrated with tubercles throughout; heart small and contracted; pericardium slightly adherent by fibrous bands; uterus normal; ovaries rough, nodulated, and congested; the veins leading to the uterus were carefully examined, and found free from disease. There were ulcerations in the ileum, cæcum, and ascending colon. Liver granular and friable; gall-bladder filled with calculi. The left iliac vein and the left femoral, as far as the popliteal space, were round, cord-like, and adhered to the adjacent structures. The cellular coat thick, condensed, and firm, so much so that it was difficult to tear it asunder. The serous coat thickened and preternaturally opaque, and the coagula were adherent to it.

Case 4.—William R—, æt. 32, admitted into the Brompton Hospital, December, 1857, in the last stage of phthisis. For fourteen days previous to his admission both feet and ankles were somewhat swollen in the evening, but the swelling subsided after a night's rest. About a week after his admission, the œdema was observed to be confined to the left foot, and to be steadily advancing up the leg. On January 8th it had extended to the groin. The whole limb was much enlarged, pitted on pressure, though slightly, and the skin was white and shining; there was also great tenderness along the course of the femoral vein, a few days before his death, which occurred on January 18th. There was congestion of the superficial veins about Poupart's ligament.

Post-mortem examination.—The right lung was riddled with cavities, running one into the other, containing a large quantity of thin, unhealthy-looking pus; a vomica of large size at the apex; pleural surfaces on right side glued together by adhesions. The leg was œdematous from the groin to the foot, and from the incisions made for the dissection of the veins a considerable quantity of serous fluid drained away. The external iliac and the femoral veins presented undoubted signs of inflammatory action. The inflammation was most violent along the inferior portion of the femoral vein. The walls of the iliac, femoral, and several of the tributaries, including the profunda, were thickened, but the thickening was much greater along the middle and inferior portion of the femoral vein than at any other part. The sheath of the vessels was intimately bound to them, forming one compact cord, with difficulty separated (even with the scalpel) into its component parts of artery, vein, and nerves. The calibre of the femoral vein was greatly increased, and the vessel filled with coagula, firmly adherent at the middle and lower part; but at the upper part of this vein, and along the external iliac, they were only partially adherent. The upper part of the external iliac, and the internal iliac, were thin, and presented no signs of disease; the femoral artery was somewhat thickened, and its calibre reduced. The nerves were so closely adherent to the sheath that it was difficult to separate them with the knife. Mucous membrane of intestines somewhat congested, but not uniformly; no ulcerations.

I am indebted to the kindness of my friend and colleague, Dr. Alison, for the particulars of the dissection of Cases 3 and 4; and in his note to me, with reference to the former, says: "I am disposed to refer the occurrence of phlebitis, in this case, to the great impurity of the blood, which could not fail to result in a special manner from the very diseased condition of two of the depurating organs of the body, the lungs and liver.

Many similar cases are on record. M. Bouillaud, in a paper in an early volume of the 'Archives de Médecine' ("De l'oblitération des veines, et de son influence sur la formation des hydropisies partielles"), relates two which occurred in phthisical subjects, and in which no mention is made of any abdominal or pelvic disease; and he quotes another from M. Breschet, in a man, "dévoré par une phthisie pulmonaire."

M. Velpeau, also, in vol. vi of the same journal, gives a case of this partial œdema, occurring in a man who died of phthisis, in which the veins in the corresponding limb were obstructed by coagula. Two instances are also recorded by Mr. Holberton, in Vol. XVI of the 'Transactions' of this Society, to which I shall subsequently refer; and another is given by Mr. Henry Lee, in Vol. I, No. 1, of the 'Proceedings' of the Society.

With reference to the cases I have recorded, I may remark that, both in the symptoms observed during life and the morbid appearances discovered after death, they correspond closely with those described under the name of phlegmasia dolens. A short analysis of them, however, will, I think, tend to show that they must have some other origin than the one to which that disease has, on high authority, been attributed; viz., the extension of inflammation by continuity of surface, from the uterine or neighbouring veins. All were individuals in the last stage of phthisis; and three of the four, it will be observed, occurred in males. The cases of Mr. Holberton, to which I have referred above, were phthisical patients, and in one (a youth) there was ulceration of the mucous membrane of the large intestines, especially the rectum. Mr. Holberton evidently inclines to the notion that the condition of the veins originated from this ulceration, and quotes the opinion of Dr. Robert Lee, that diseased structure may give rise to inflammation of the neighbouring veins; and also refers to a case cited by Dr. Lee, of a man who died of cancer of the rectum, in whom Mr. Lawrence found the iliac veins inflamed and obstructed.

Now, whatever may have been the case in the above instances, the disease could not have had this origin in Cases 2 and 4, as in neither was there any ulceration of the intestines, nor any diseased structure in the neighbouring parts, from which the disease could have been transmitted by continuity of surface.

It appears to me they come under the class of cases described by Mr. Henry Lee, in which coagulation in the vein is produced by the presence of pus or some foreign matter in the blood; and that the subsequent inflammation was owing to the stagnation of vitiated blood in the vessels. All were in the last stage of phthisis. The lungs contained numerous suppurating surfaces, from which pus or some foreign matter might readily be conveyed into the circulation.

With reference to this view of the question, the following remarks, made a long time ago by an acute observer, may find their application. "Si, dans beaucoup de cas, la cause première des maladies réside dans les solides, elle peut ensuite être transmise aux fluides qui réagissent à leur tour sur les solides, en vertu du consensus étroit qui existe entre les uns et les autres. Or, combien de produits morbides formés dans le sein de nos organes peut ainsi servir de germes à de nouvelles maladies; car toute matière purulente en contact avec des tissus vivans, toute désorganisation qui tend au ramollissement, sont des espèces des poisons intérieurs plus ou moins actifs dont l'absorption peut s'emparer. N'est-ce pas ainsi qu'il faut expliquer ces fièvres consomptives qui accompagnent les grands foyers de suppuration, le ramollissement des tubercules, du cancer, et plus tard ces lésions nombreuses, et ordinairement de même nature, qui surviennent dans une foule d'organes, comme autant de rejetons de la première maladie?" (Dance, "Sur la Phlébite," 'Archives de Médecine,' vol. xix.)

CASE

OF

RECURRENT ANÆSTHESIA

OF

ALMOST THE ENTIRE SURFACE OF THE BODY,

ACCOMPANIED BY PARTIAL LOSS OF MOTOR POWER IN ALL THE LIMBS, TRACEABLE TO THE EFFECTS UPON THE SPINAL NERVES OF EFFUSED PRODUCTS WITHIN THE SPINAL CANAL.

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The accompanying case I have thought worthy of being submitted to the attention of the Society, chiefly for the following reasons:

1st. On account of the great infrequency of the post-mortem appearances met with.

2d. On account of the character, cause, and mode of access, &c., of the various symptoms.

3d. On account of the close and intelligible dependence of the symptoms upon the lesion, as determined after death.

Previous history.—The patient was a tall, well-made bricklayer, who had been in the habit of drinking as much as fifteen or sixteen pints of "half-and-half" daily, and when at work several pints of gin in addition. About six years before his fatal illness, in the winter, and whilst at work building (being otherwise in good health), he became, for the first time, affected by numbness and the peculiar sensation usually termed "pins and needles" in his lower limbs. He, however, continued his work for some weeks, experiencing no other discomfort whatever. Becoming at last unable to follow his avocation, he was treated by a medical man; and, under the use of repeated blisters to the back, in the space of about four months "quite recovered."

After this first attack he was for some weeks "every winter" affected (though to a less degree) by the same kind of numbness and peculiar sensations, but was never compelled to give up his work until the beginning of the winter of 1852-53, when the old symptoms returned, but with greater intensity, and affecting the arms as well as the legs, and at this time he came into St. George's Hospital.

Symptoms.—When admitted he appeared to be enjoying good health and spirits, and was quite "free from pain." There was, however, the following interference with motor power, and general sensibility of the integuments. The sensibility to pinching or pricking of the skin of either leg, as high up as the middle of the thigh, was extremely slight, very sharp and continued pinching with the fingers being only faintly perceived. A similar kind and degree of obtuseness as regards pinching and pricking of the skin of both arms, at all parts below the elbows, was also found to exist, and both cheeks, as well as both sides of the nose, were in like manner very deficient in sensibility.

The tactile sensibility of the skin of the forehead, as of all parts of the body not alluded to above, was quite natural.

As regards voluntary motor power, the muscles of both arms and hands were manifestly much weakened, the grasp with both hands being extremely feeble and limited. The muscles of the legs were also much weakened, as the patient could only walk with crutches and with a tottering gait, and was unable to stand unsupported. The sphincters did not seem to be affected. The mental powers appeared to be perfectly natural, and all the special senses quite perfect; but the patient complained of restlessness and want of sleep, and of troublesome dreams.

Slight involuntary twitchings of all the limbs were at times noticeable.

No pain or tenderness could be induced by pressure upon, or movement of the head or spinal column. The pulse was regular; the appetite good; the bowels somewhat inclined to constipation; the urine was high coloured, and, although at one time only having a specific gravity of 1011, was free from albumen. The specific gravity subsequently much increased. Auscultation and percussion indicated nothing wrong in the thoracic organs.

The patient was treated by repeated blisters to the spine, by diuretic medicines containing the nitrate of potass and tincture of cantharides, and by small doses of mercury. The use of the cantharides was pushed so far as to produce decided irritation of the kidneys; for this reason it was three or four times omitted and resumed. This line of treatment was continued with short intervals for above four months; at one period the extract of nux vomica being resorted to, whilst the cantharides was left off.

After a time some, but not much pain was complained of in the hands, feet, and thighs; and occasionally there was pain across the abdomen; but no pain was complained of either in the head or back. Unusual severity of cold weather was wont to increase the numbness in the limbs, but he recovered gradually so far as to use his limbs pretty freely, and to experience but a slight amount of numbness, and that only occasionally. He then left the hospital, and about two months after doing so the numbness of both hands, along with partial loss of power in them, recurred, but especially in the right one. He continued taking the diuretic medicine with the cantharides, and in a week or two the symptoms again departed.

Five months later, numbness of both arms and hands recurred, appearing firstly and mainly in the right side, with partial loss of power; and somewhat later numbness in the left foot also. These symptoms came on, and again went away several times. Slight pain in the head also supervened, not limited to any region; but there was no giddiness, and apparently no confusion or aberration of thought. Under the continued use of the tincture of cantharides and small doses of strychnia he again recovered, and left the hospital free from any numbness, and with perfect sensibility of the skin. After this nothing is known of the condition of the patient for the term of a year and a half; at the end of which period he was brought a third time into St. George's Hospital, but on this occasion in a very soporose condition, rambling in mind when spoken to, and with slight trembling of the facial muscles. The tongue was furred and dry, and the pulse quick, 100 per minute, and feeble, but regular; and it was at first thought that he was the subject of continued fever.

Owing to his mental condition, it was very difficult to make out to what extent the powers of motion or the sensibility of the skin were interfered with. There was, at any rate, no actual paralysis or spasm of the muscles of the face or eyeballs, and the pupils acted naturally. There was no vomiting.

He was cupped at the back of the neck, and freely purged. During the night he was rambling much, and continued in a semi-comatose state, secreting spoons and other objects under the bed-clothes. There was occasional twitching of the muscles of the arms and legs.

On the following morning the pupils of both eyes were noticed as being "contracted." The stupor continued, and great irritability of temper came on, manifested by the fits of anger which he gave way to whenever any part of the body was even slightly pinched. During the next night he had some kind of a "fit," attended by much struggling. This was followed by dyspnœa accompanied by much noise in the throat, and by death on the third day after his final admission into the hospital.

Post-mortem examination sixteen hours after death.—The body was well made, and muscular in all its parts. No emaciation existed. All the limbs were rigid, the thumb of the left hand being firmly flexed into the palm.

The heart was quite natural, but the lungs, kidneys, liver, and spleen, and also the lower part of the small intestines, were highly congested.

On examining the *"head,"* the cranial bones were found to be quite healthy. The cerebral membranes at the upper parts of the brain were natural, but the surfaces of the convolutions were dry and flattened. At the central part of the base of the brain, posterior to the optic commissure, much tolerably firm fibrinous material existed beneath the arachnoïd membrane, matting together the various vessels and nerves of the part. The pituitary body was very indurated, being infiltrated with and surrounded by similar fibrinous material. The brain-texture itself was natural, excepting very slight softening of the boundaries of the lateral ventricles, which were full of a quantity of limpid fluid.

On examining the *"spinal column,"* the vertebræ, as well as the dura mater covering the spinal cord, were found to be natural. The spinal arachnoïd membrane was, however, more than usually opaque; and here and there, although infrequently, bands of adhesion existed between this membrane and the subjacent pia-mater, along various parts of the spinal cord. The most interesting changes observed were in connexion with several of the spinal nerves. In many parts, though chiefly at the lower cervical and the upper dorsal regions of the cord, both the anterior and the posterior roots of the nerves were invested by very firm fibrinous material, partly of a white and partly of a pinkish colour. In many cases this deposit was so scanty as only to be seen on close inspection, coating over the nerve-roots. In other places the deposit was sufficient to form a layer, obscuring and matting together the various nervous strands composing the entire nerve-root; whilst in a few cases the deposit existed around the separate roots in single masses, appearing like beads of various sizes.[1] In one instance (see Plate XI) it formed a pink-stained mass around one of the anterior roots on the right side, of the size of an ordinary pea, and having (except for its colour) much the appearance of a neuroma or gangliform enlargement. The spinal cord itself was natural, both as regards size, colour, and consistency.

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[1] The preparation of the spinal cord will be placed in St. George's Hospital Pathological Museum, as No. 5, Sub-series iv, Series xxiv.

On microscopical examination, I found that the great part of the material investing the nerve-roots possessed all the histological appearances of ordinary fibrinous material; but, on examining that portion of the exudation which immediately surrounded and penetrated between the various nervous elements forming the entire root, in addition to a quantity of granular and amorphous material, there were found vast numbers of peculiar round and oval, and in some cases oat-shaped, bodies. On the application of acetic acid, these bodies became somewhat larger (see Plate XI, fig. 2), but did not appear to alter in any other way. The nerve-fibres of the various affected nerve-roots were, in most places, very opaque, and filled with granular matter and fatty particles and globules. Where the masses about the nerves were of a pink colour, blood-globules were found mixed with the material.

Remarks.—In reviewing the various phenomena of the above case, I will limit myself strictly to the points which appear to me mainly worthy of consideration. I will, in the first place, allude to those morbid changes discovered after death which more obviously deserve notice, that is, to the pathological condition of the roots of the spinal nerves.

The adventitious material attached to, and in many places surrounding firmly, and as it were incorporated with, the roots of the nerves, was manifestly the remains of some exudative process which at some time or another had, during life, affected the spinal membranes. Whatever may have been the precise nature of this process, whether, that is to say, it was one of a purely local nature, and of a kind usually designated inflammation of the spinal membranes (the masses of deposit about the nerves being, in fact, only part of some exudation, the rest, chiefly fluid having been absorbed), or whether it was of a more unlimited nature, an expression or manifestation, *i. e.*, of some general cachectic state such as will induce, as we know, fibrinous effusions simultaneously into various organs and upon various free surfaces,[1] it was equally interesting and important to find that the chief results of the exudative processes had become aggregated around the roots of the spinal nerves.[2] In this way, no doubt, these roots had become much compressed, being in a degree affected much the same as if a ligature had been passed round them. In not any of the cases of acute or chronic spinal meningitis which I have either met with or heard of, has the inflammatory exudation become so disposed as in the present case.

Then again as regards the chief symptoms manifested during life, the tendency to their recurrence, with intervals of perfect freedom, cannot fail at once to strike the mind as peculiar. Thus, we find the patient first of all affected in his lower limbs, as to motion and sensibility, for a period of five months; then for a time enjoying entire immunity; then being subject to slight relapses every year, but only in the winter-time; then having a relapse so severe as to necessitate discontinuance of his work; then, after partial recovery, having slight attacks, the upper limbs only (and chiefly the right one) being affected; and finally, after several more slight invasions, being attacked by his last illness, the commencement of which we are quite ignorant of, but which differed materially from all previous attacks.

As far as I know, this recurrent character of the symptoms is very unusual; for although in certain cases of spinal meningitis, in which spasm is produced, painful convulsive action of muscles is wont to appear irregularly, and with remissions, yet it is rare to have such a chronic series of attacks with lengthened intervals as in the present instance.

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[1] As regards the predisposing cause of the exudation, perhaps the generally good condition of the patient, and the exciting cause of the attacks (outward cold), would be opposed to the idea of any cachectic influence having been at work; whilst, on the other hand, his intemperate habits favour such a view.

[2] Possibly the accumulation of the adventitious material into mostly rounded masses may have been partly owing to the pulsating movements which the spinal membranes, and perhaps also the cord itself, undergo during respiration.

Respective of the course which the symptoms took, it appears very clear that the nerves of the lower part of the cord were first in order affected, then those of the upper part, and finally the fifth cranial nerves, owing to the disease travelling gradually upwards from the lower part of the spinal to the base of the cranial cavity.

It is to be observed that, except towards the last stages of the disease, little or no pain was at all complained of. This was most interesting, as pointing rather to the immunity from disease enjoyed by the spinal cord itself; but it was certainly unusual to find that such alterations in the spinal membranes and nerves had occurred without more pain having been experienced, and with so little spasm or rigidity of muscles; the only tendency in this direction noticeable being the slight twitchings of the various muscles. This continued freedom from pain or pathological muscular contraction should, no doubt, be attributed to the extreme slowness of the diseased action.

Later on, however, we have the history of pain in the limbs, and slightly in the abdomen; this may have been owing to the supervention of some alteration in the nerve-fibres, as we see from microscopical research that they had become themselves affected structurely. And as time progressed, the mind, which had hitherto remained unaffected, became much disturbed. This accession to the symptoms might well be set down to secondary changes coming on in the brain, and, no doubt, corresponded with the invasion of the sub-arachnoïd tissues at its base, and with the softening of the walls of the ventricles, with effusion into their cavities.

I will not fail to notice the numbness of the cheeks and nose. This, doubtless, resulted from some implication of parts of the fifth cranial nerves, either from softening of their structure or compression by effused products; but how it came to pass, considering the position of the intra-cranial effusion, that these nerves should be involved, and yet, as it seemed, the third and fourth nerves escaped, whilst after death all appeared to be alike implicated, I cannot explain. I can only, by way of illustration, call to mind the fact that very frequently extensive sub-arachnoïd effusion of lymph, in scrofulous meningitis for example, is found at the base of the brain, matting together the various nerves, and yet no special symptoms referable to the muscles supplied by them are produced. Before closing, I would draw attention to the microscopical appearances depicted in the Plate accompanying the case. The nerves, as before described, are seen in a very unnatural condition, being opaque and granular, and containing much fat in the form of globules. This alteration in the roots of the nerves which were affected by the adventitious material I am inclined to attribute mainly to the pressure of that substance, although possibly the roots of the nerves may themselves have been affected directly as to their nutrition, by virtue of the diseased process extending to them from the investing serous membrane.[1]

The histological character of the deposit found amidst the various nerve-fibres of the affected roots is peculiar, and is well seen in the Plate XI, fig. 2. I do not remember ever to have met with exactly similar structures as the small corpuscles present in any exudative material that I have examined.

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[1] Such extension of diseased action from membranous surfaces to subjacent structures is well seen, for example, in certain cases of pericarditis, where the muscular substance of the heart becomes affected consecutive to the affection of the pericardium.

EXPLANATION OF THE PLATE.

The drawing represents a case of enlargement and cohesion of the roots of the spinal nerves, anterior and posterior.

Fig. 1 shows a portion of the lower part of the cervical region, of about the natural size.

Fig. 2 shows the microscopical appearances of some of the affected nerve-roots, along with that of the adventitious material.

Plate XI

Fig. 1.

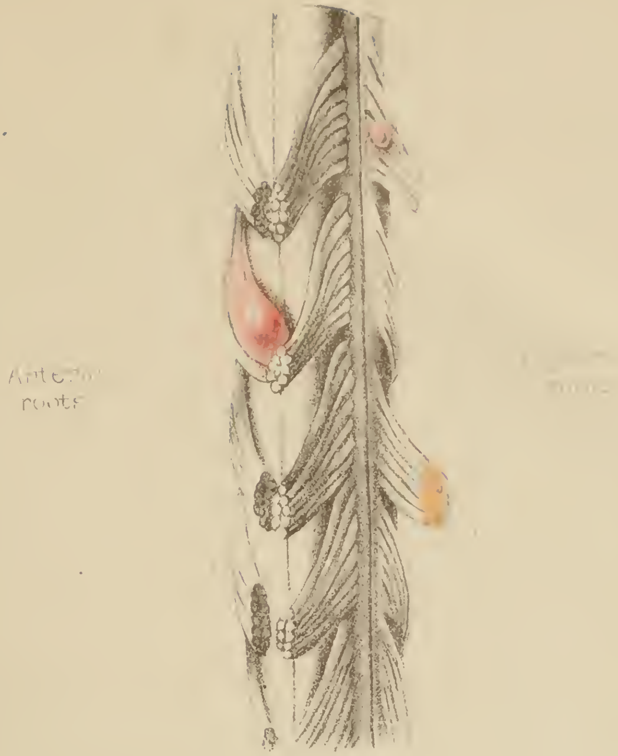
Anterior roots
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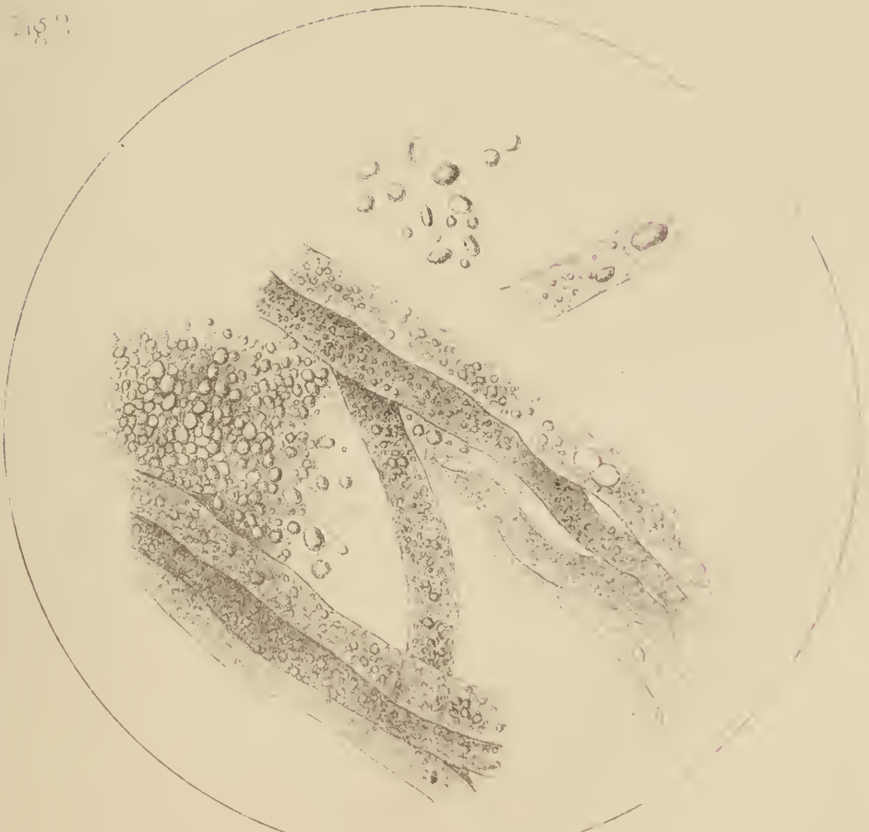
Fig. 2.



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G.H. Ford chr lith.

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